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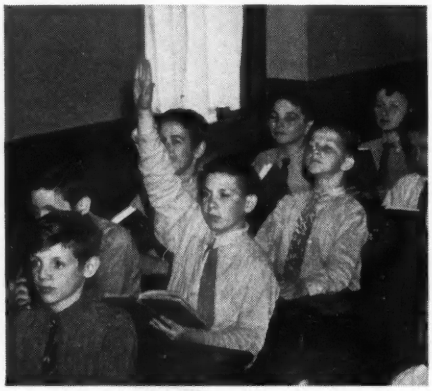
LUME 78 NUMBER 3

"... vitamin B_{12} does exert a 'growth-promoting effect' when given as a dietary supplement ... this evidence is exceptionally strong."[1]



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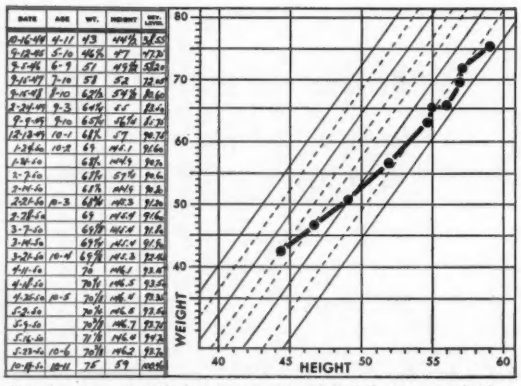
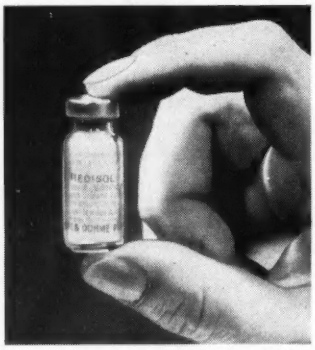


Growth pattern of child plotted on the Wetzel Grid shows unmistakable progress during the 4-month period of vitamin B_{12} supplementation.



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Subtotal Gastric Resection for Peptic Ulcer

Preliminary Report of a Variation in Technique

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• Internists as well as surgeons agree that subtotal gastric resection is a satisfactory method of treatment for a selected group of patients with intractable or complicated peptic ulcer.

A short historical review of the development of the operation is given.

The importance of removing a large portion of the acid pepsin-secreting area of the stomach is stressed. A variation from the usual method of resection accomplishes this and at the same time leaves a satisfactory gastric pouch and lessens the incidence of the dumping syndrome.

INTERNISTS AS WELL as surgeons agree that subtotal gastric resection is a satisfactory therapeutic measure for a selected group of patients with intractable or complicated peptic ulcer. That it is not accepted as a final solution to the problem is obvious and surgeons are constantly striving to improve the operation technically and physiologically until such time as other and better methods of treatment will have eliminated the need for surgical treatment.

So much has been written on gastric resection since publication of a report on Billroth's[2] first successful operation in 1881 that it would now be impossible for a practicing surgeon to review personally all the papers on this subject. However, even occasional partial review should help toward fuller understanding of the development of and the reasons for present procedures, and thereby provide a basis for correct evaluation of new procedures as they appear.

Resection of from three-fourths to five-sixths of the distal portion of the stomach for peptic ulcer has come into general use because removal of a lesser amount of stomach, as was done formerly, was followed in a high proportion of cases by recurrent gastrojejunal ulceration. The early method of resection of the pyloric antrum alone was based on Edkins'[13] observation that this portion of the stomach when stimulated secretes a hormone, gastrin, which activates the secretion of hydrochloric acid.

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It soon became evident from the clinical experience of many surgeons, however, that removal of the antrum alone was not enough and that in addition removal of a part of the body of the stomach with its acid-secreting cells was necessary if permanent cure was to be attained and secondary jejunal ulceration be prevented.

The more extensive resection entailed greater incidence of disagreeable sequelae, particularly the dumping syndrome. While the exact cause and nature of this syndrome is not understood, it seems reasonable to assume that physiological and mechanical factors resulting from the loss of so large a portion of stomach play a role.

Connell[3-10] introduced in 1929 a new concept in the principle of the surgical treatment of ulcer.

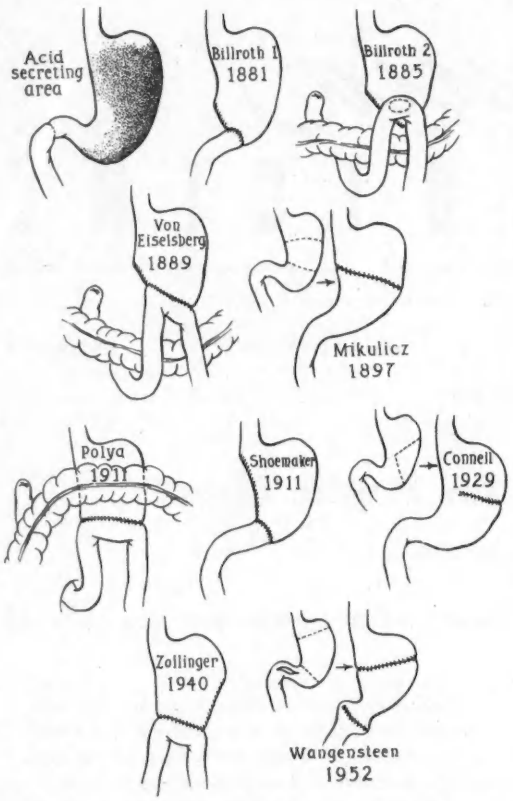

Figure 1.—Location of acid-secreting cells in stomach and methods of subtotal gastric resection.

Accepting the importance of the acid pepsin factor in the production of ulcer, he recommended removing a large portion of the acid-secreting area and at the same time retaining the alkaline-secreting antrum and the pyloric sphincter. He did this by making a wedge-shaped excision of the body of the stomach with the apex of the wedge close to the lesser curvature but not dividing it. He recognized that this could not be done as an isolated procedure in the presence of obstruction, but advocated and himself performed plastic procedures at the pylorus where this was indicated. Before performing the operation on selected patients he satisfied himself of its feasibility by carefully controlled experiments upon animals. The series of clinical cases which he reported, while not large, indicated good results following the operation. In experiments by Connell and by other investigators it was observed that the acid values in fundusectomized dogs, after an initial decrease, soon returned to preoperative levels. Mann[16] pointed out that an estimate of the volume of secretion rather than of acid value alone might be a better method of determining the possible value of the procedure. Connell's method of fundusectomy has not been taken up by other surgeons, largely because the experimental work does not seem to justify it and because of the good results obtained by adequate and properly performed subtotal gastric resection by more orthodox methods. That his conclusions have not been widely accepted is not sufficient reason for ignoring them, and it may well be that his deductions should be made the object of further experimental work and studies to see if they may not at least in part be incorporated into the present-day concept of the surgical treatment of peptic ulcer.

More recently Wangensteen[24, 25, 26] has adopted Connell's concept of the desirability of removing the acid-secreting area of the stomach and leaving the gastrin-secreting area. He accomplishes this by extending the old segmental resection of Mikulicz[17] which removed a relatively small section of the body of the stomach. Wangensteen removes almost all the body of the stomach, leaving the pyloric antrum and a small portion of the fundus, which are then united in continuity. To overcome the effects of the resulting interference in motility he widens the pylorus by means of an extensive Heineke-Mikulicz procedure. Wangensteen reported good results.

Like Connell's, Wangensteen's operation has not been accepted by the surgical profession and for basically the same reason, namely, that a surgical procedure like the orthodox subtotal gastric resection, while admittedly not perfect, has stood the test of time and will not be superseded until another procedure has been proven superior by the same demanding tests.

In 1940 Zollinger[27] combined so-called fundusectomy with pyloro-antral resection. He did this by removing the body of the stomach and the greater curvature well up to the fundus and reestablishing continuity by anastomosing the jejunum to the remaining gastric pouch adjacent to the lesser curvature. The operation was almost total resection and was used only in a limited number of complicated cases.

Many methods of reestablishing gastrointestinal continuity after partial gastric resection have been described. Basically they are all modifications of either of the two methods originally described by Billroth, namely:

I. Direct anastomosis between the proximal and distal segments.
II. Closure of the distal segment and anastomosis of the proximal segment to the intestine somewhere below the closed end.

There is no need to review again the many types of operations described to accomplish this union. Accepting the present view of the importance of the acid pepsin factor in the cause of ulcer it can be said that success of an operation depends more on

removal of a sufficient amount of stomach, including a portion of the body together with the pylorus, than it does on the particular method in which this is accomplished. The author considers a short loop desirable and therefore makes the anastomosis beneath the transverse colon, suturing the edges of the opening in the mesocolon to the stomach wall above the line of anastomosis. Occasionally if anatomical conditions favor it, a Billroth I operation is done. No exact way to measure or to designate how much of the stomach is removed has been established. Fractional terms such as "three-quarter resection" and "seven-eighths resection" are used by surgeons, but it is probable they are inaccurate and often indicate resection of a larger area than was actually removed. It cannot be seriously questioned that the larger the portion of the stomach removed, the greater are the technical difficulties for the surgeon and the greater the risk and discomfort for the patient.

MODIFIED TECHNIQUE

These considerations have led the author to a modification of the usual technique of gastric resection which meets the requirement of removal of an adequate portion of the acid-secreting area of the stomach yet retains a little more of the lesser curvature or magenstrasse which is important for the motor function of the stomach. It is essentially Zollinger's operation done in a somewhat less radical manner.

The operations commonly used today are the Polya[19] and Von Eiselsberg[22] and Schoemaker[20] modifications of the Billroth II procedure. In the Polya operation the whole circumference of the stomach is anastomosed to the side of the jejunum, whereas in the Von Eiselsberg and Schoemaker modifications only that portion of the resected stomach which meets the greater curvature is used for the anastomosis. The resection is made to include most of the lesser curvature and a large part of the greater curvature and fundus remain undisturbed.

Cox[11] and other investigators[1, 12, 18] showed that the acid-secreting cells of the stomach are concentrated in the body of the organ and along the greater curvature well into the pyloric zones and that they are less in number in the fundus and along the lesser curvature. This being so, it would seem desirable to resect a larger portion of the stomach along its greater curvature with its acid-secreting cells and to retain more of the lesser curvature. The operation used by the author accomplishes this. Because of the large mucosal folds in the body of the stomach and along the greater curvature, this procedure will remove considerably more acid-secreting area than a measurement of the corresponding serosal surface would indicate. The lesser curvature is freed to about

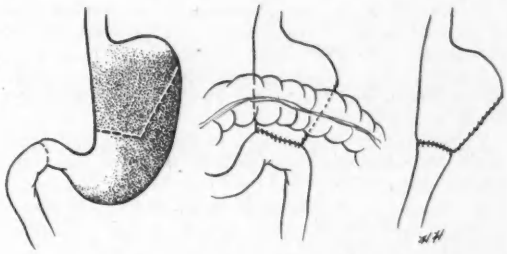

Figure 2.—Illustrating operation in which excision of a large part of the greater curvature removes relatively more acid-secreting cells yet retains relatively large gastric remnant.

the junction of its upper and middle thirds and a clamp is placed at right angles to it to include a sufficient amount of the stomach for satisfactory anastomosis. A second clamp is then applied at an angle along the greater curvature so as to remove the desired amount of this part of the stomach. The portion of stomach included in this clamp is then closed and the anastomosis made to the stomach adjacent to the lesser curvature either as a Billroth I or Billroth II procedure. The author has done the operation in enough cases to know that the stomach pouch functions well after operation. Further studies, now in progress, may help toward better evaluation of the procedure.

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A.M.A. Revises the "Essentials of an Approved Internship"

AN ADVISORY COMMITTEE ON INTERNSHIP, appointed by the Council on Medical Education and Hospitals in the fall of 1951, conducted a study in the past year reviewing the internship in its broadest aspects. As a result of its study the Advisory Committee recommended revisions in the "Essentials of an Approved Internship" which were ratified by the A.M.A.'s House of Delegates in December.

Among the changes in the requirements for hospitals offering intern programs were the following: Approval by the Joint Commission on Accreditation of Hospitals; bed capacity increased to 150, excluding bassinets; annual admissions increased to 5,000, exclusive of the newborn, and the autopsy rate increased to 25 per cent.

Under these revisions the Council will approve rotating and mixed internships and straight internships in these specialties—internal medicine, pediatrics and surgery. Straight internships in pathology and obstetrics-gynecology will no longer be approved.

The revised "Essentials" became effective January 1 for new approvals. The autopsy rate of 25 per cent became effective for all hospitals January 1.

—A.M.A. *News Notes*

Preventable Diseases: The Scope of Public Health

HAVEN EMERSON, M.D., *New York, N.Y.*

• The purpose of a department of public health is to put into effect for the benefit of all the people of a community the practical lessons of preventive medicine. The scope of public health work is chiefly determined by our knowledge of the causes of the preventable diseases. We do not know how to prevent all diseases. We do, however, know effective ways to prevent or to reduce the occurrence of at least a dozen varieties of disease.

Every health department ought to provide for a program and services which will prevent so far as possible the preventable diseases and the preventable hazards to health. To concentrate on prevention is to avoid diversion by specious arguments into the fields of medical care of the sick.

IT HAS SLOWLY PENETRATED the minds of the devotees, the disciples, and the crusaders for public health that science and democracy are a team that cannot be managed from the bleachers. Whether we are physicians, engineers, nurses or any of the half dozen other associated professionals concerned with public health work, we have to get into the game ourselves and play ball, whether in the sand lot or on the turf of a levelled diamond.

We are deeply concerned with a true and enduring marriage between medical science, or human biology, and society, that is, our own local community and its elected and appointed officers of government.

Without reviewing the experience of the past century, we can, I think, agree that there is a close similarity in the duties and organizations of local health departments across our continent.

Granted that the scope of public health is as broad as human biology, with its subtopics of sociology, eugenics and ecology, there is still place in our thinking for priority, for emphasis, for timeliness and for exercise of discrimination in the conduct of our public service.

The sick we shall always have with us, and we can be assured that leadership and responsibility for the care of them will be the major undertaking of the medical profession in the foreseeable future. We, the minority group concerned with the health of the public rather than with the care of its sickness, can claim a certain distinction in that we seek goals unthought of by the great mass of patients and physicians who live in the hope of a miracle drug or the flashing knife that searches ever deeper into the very brain and heart of man.

What is in fact our chief preoccupation, that glint in the eye, that lift of the chin that marks us as fanatics by choice? We have, I think, forsaken the ancient gods, the temples of Aesculapius, for new sanctuaries where we worship the cult of prevention. We can imagine ourselves a sort of priesthood that can ward off all kinds of sickness and ill health. Our belief, our hope, our career is in prevention.

There is, of course, no calculable end to the list of preventable diseases. It is added to almost daily, and with the increasing length of the list we see the weight of fear and anxiety, ignorance and superstition, uncertainty and despair fall from the shoulders of fellow beings, from the centenarian to the babe unborn.

The earliest and still, to my mind, the prime object of the sciences of preventive medicine is the sparing of man and his society the suffering, injury and shortened lives due to preventable diseases.

I solicit your criticism, your objection to my tentative categories, your outspoken suggestion of omissions, as well as of my errors of commission. I offer you as a working basis, more or less in the order of social or numerical importance, a listing of what I believe to be the preventable diseases. Until we have done our utmost to prevent what we know to be preventable, I see little excuse for our engaging the army that parades under the flag of public health in the care of the sick. Second to our obligation to make effective use of what we believe we know about preventing disease is a duty to use the formidable resources of demography, epidemiology, biostatistics, biochemistry, genetics and the whole range of experimental techniques to discover clues to preventability of diseases still thought of as unavoidable, and then apply our new knowledge in the field to practical purposes, to social ends.

First in biological and numerical importance are diseases of nutrition. While gluttony, the cause of most obesity in the United States, marks some 25 to

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30 per cent of us as suffering from surfeit, more than half the world's people face each day the probability of insufficient food to permit growth, development, repair resistance to infection. We can be as precise and definite in describing the deficits of diets, the results of general and specific shortages or lack of nutrient and accessory food factors as we have been in epitomizing our knowledge of the communicable diseases.

While there are broad gaps in our information it must be said that we do now know enough to prevent a long list of preventable disorders of nutrition or metabolism. Not by authority of law in most instances but by the persuasive power of education and the resourcefulness of agriculture, transport, chemistry and economics, we can do more than we have yet imagined. There is a limit to the diseases of nutrition.

Second in importance, it seems to me, are the occupational diseases, all of which are preventable.

Of these there is no end. There is no occupation but compulsory military service that must be tolerated in the public interest if the employment hazards the worker's health. War may be considered a form of preventable mental disease.

This whole field awaits the health officer, for surely no one else is more than picking at it. Not labor, not management, not the surgeon, toxicologist or engineer alone, but all together are needed for prevention. I see it as a public duty for the health officer to know the places and conditions of work of all occupied people in his community, at least those self-supporting persons working outside the home. For lack of morbidity reporting, and of records of absenteeism from work and its causes, we live as it were with statistical blinders on our minds, waiting some dramatic episode to reveal a new kind of glue, plastic, dust, fume or fatigue to bring a workman to death's door.

Environmental sanitation of the future will be less concerned with smells and smoke, with pigpens and winery wastes than with the places and conditions where men and women work, and the results of these upon their lives as well as upon their livelihoods. We shall see on the walls of health department offices pin maps designating occupational hazards, reported cases of inadequate space, light, cleanliness of air and other factors leading to illness or disability. We shall be as determined to stop epidemics of lead poisoning as to control smallpox. When men and women leave their homes to work indoors or out, they will be conscious of the concern of the local health department with their shop, office, ship or plant.

Third in current importance in our own country is the category of communicable diseases, the conquest of which has been the main purpose of health organizations around the world. Our work is popularly measured by our successes and failures in this field. We can more nearly honestly speak of control of these than of any other kind of preventable disease. Perhaps the greatest triumphs of the future will be the hoped-for mastery of the common cold, the dysenteries, gonorrhea and neutropic diseases of virus origin. In no other field of preventable diseases does the performance of public health services come so near to applying effectively the sciences which have put in our hands the resources of diagnosis, immunization and specific or selective therapy. We can comprehend if not finally list and classify the communicable diseases in their entirety. Doubtless new and unexpected infectious vectors, mutants of existing or developing microorganisms and their transmittors in the insect and animal kingdoms, will arise to plague us anew, but our resources are many and we can await such biological novelties with confidence in our past experience.

The fourth group, and perhaps the most important in terms of international as well as personal health, is preventable conditions of the spirit of man. Disturbances and defects of mentality, personality, emotions, behavior or interpersonal relationships now begin to receive the attention they warrant as we shift emphasis from concern chiefly with institutional care of the already mentally sick or socially inadequate to guidance by sympathetic education in the natural history of mental health. No more difficult task has faced the health officer than the obligation to interpret to his community, the families, school teachers and children, ministers, publicists, legislators and public servants, the principles of character development, emotional growth and the basic types of human conduct that, if ignored, may lead to such despair and frustration of life as no other of the ills of man approach. We fumble with the facts, struggle with the structure of administrative application of the truths already manifest to us and grope for methods of approach to the motives of man upon which his often disordered conduct depends.

No more difficult problem faces the sciences of preventive medicine and yet none discloses and demands more varied forms of cooperative and interprofessional concern. If health is to be the happiness we believe it can be, and not merely a dull normality of bodily functions, an absence of physical pain or fever, the mind and spirit of man must be released from the handicaps that have developed under the restraints, the traditions, the urges to dominate, the social complexities of our day.

Fifth in order is one of the closely related phases of the last described, namely, the effects on body and mind of the habit-forming drugs, primarily the derivatives of opium, coca, cannabis and, the most prevalent and destructive of all, ethyl alcohol. Some would add tobacco, peyote and marihuana. It seems to me that of all the glaring, insidious, persistent and deteriorating practices of man, the voluntary drugging of his brain and central nervous system by alcohol is hardly second to any other wholly preventable condition. Well informed leaders in public health at national, state, and local levels have given well documented evidence that in terms of sickness and death, economic loss and cost to the person, the family and the community, the drinking of alcoholic beverages is numerically among the first four, at least, of the enemies of good health in the United States, with our national capital recording a higher per capita alcohol consumption than any of the states. And yet hardly a beginning has been made in any state or city to do more than provide humane care, medical skills, and a gesture of rehabilitation for persons with advanced cases of chronic alcoholism. There is no clearer challenge to the cohorts of public health than to apply themselves to education, by example as well as by precept, for the prevention of alcoholism.

The sixth category of preventable conditions is one of the tempting and intriguing, the most constructive and welcome of the established fields of public health effort—the prevention of conditions which may lead to disturbances of growth and development in maternity, infancy and childhood. Is there any other aspect of existence, or of the processes of biological continuity and evolution more precious or significant than that of human reproduction, the succession of the generations, the replacement of parents by their children, the inheritance of characteristics, the infinite variations in the genetic pattern of individuals? We are deeply concerned with each stage in the processes of choosing a mate, of being fit for marriage, for procreating, for pregnancy, and childbearing and rearing, and for the safety of the developing embryo, the neonatal period of infancy and the growth to and through school age, adolescence, employment, and around the circle until a new family is in process.

And we begin to learn latterly that we have created a problem of longevity by the very fact of saving the lives of little children and youth to grow up and grow old.

To survive until reproduction has been achieved is the minimal objective of human existence. To live the full span of life in health is a further social goal and we now find it desirable to guard the development of age as we did that of youth. I think of this field of public health effort as that of preventable conditions of development in the stages of reproduction, growth, maturity and old age. We cannot escape official concern with both fertility and sterility.

This leads to a seventh category, the preventable conditions of inheritance. We begin bit by bit to understand the skein of the chromosomes, to relate inherited peculiarities of body form and structure to particular genes. We separate better the preventable causes of congenital defects from the still unknown hereditary factors we see to be uncontrollable. True it is that our information is scanty and our social resources to prevent serious hereditary defects slender and rarely applied, but a glance through the rapidly growing library of books on inheritable defects opens a wide door of future possibilities.

Nevertheless, there have been remarkable results from systematic social and medical efforts to eradicate some hereditary blights such as Huntington's chorea, now non-existent in Minnesota.

An eighth group of disabilities, largely of inheritable origin but subject to many resources of prevention, is the protein susceptibilities, the allergies—*strange maladies*, as they have been called. Environmental protection, guided and selective personal hygiene measures, avoidance of occupational excitants, and a portion of wise medical management of personality reactions to the stresses of life—these and other resources can be helpful not only to the individual but, through mass measures and education, to whole communities such as live under the seasonal cloud of ragweed and other prevalent irritating pollens.

We now come to the last four of my groups of preventable diseases, each of which has been busily promoted by widespread social concern, research developments and some promising measures of public health administration. I refer to malignant tumors, diseases of the heart and blood vessels, dental caries, and accidents.

As to malignant tumors, we have confused the public by using the word *control* when this can be applied only to those of occupational origin, and so properly within the field of public health. Some cancer is preventable, but early diagnosis and appropriate treatment while postponing or even preventing death is not the prevention of cancer. Education of the public by the health department and the medical profession in the wisdom of early and repeated medical examinations and wide publicity as to the value of prompt treatment and how and where and by whom it can be had—these are valued promotional efforts. However, evidence of substantial results from

efforts to prevent cancer is slight, although surgical treatment and radiation have saved many persons from premature death. We await hopefully for some suggestion of preventable factors of causation, other than in certain occupations, before we can plan in entire honesty for prevention.

As for diseases of the heart, our tenth group of preventable diseases, with the exception of those due to nutritional disorders (beri beri, endemic goitre, obesity), and to communicable disease (syphilis) already included under groups 1 and 3, we have the sequelae of rheumatic fever and senescent or arteriosclerotic heart disease. It remains to be seen whether any measures intended to prevent rheumatic carditis or to delay or postpone the vascular changes of senescence actually produce any statistically significant results. At least at present the most promising factors for the prevention of the great mass of heart disease are the care of a competent physician, the avoidance of streptococcus infections and the maintenance of moderation in physical and emotional aspects of personal hygiene. The publicity and popular promotion of interest in heart disease as the most frequent of the causes of death may well be justified for fund-raising purposes and to encourage people to have periodic health examinations at suitable intervals, but evidence of a reliable kind is lacking to support claims that the prevention of heart disease has made any appreciable progress unless it be in rheumatic carditis and deaths from this condition in persons under 30 years of age.

Dental caries is the eleventh category of preventable disease and there might be added other defects of dental development. Despite the fact that we do not know the main or underlying cause or causes of dental caries as it prevails among preschool and school age children, there are at least three measures that have brought about very substantial (about 45 per cent) reduction in this condition: Oral hygiene supplemented by prophylactic cleansing of teeth by dentists; diets such as the Oslo diet and the supplementation of a balanced diet for children by vitamin dosage, and combined with omission of or great reduction of refined sugar use in foods and soft drinks; and the use of fluorides either by topical application to the teeth or by fluoridation of drinking water supplies. Perfect methods and results have not been attained but great benefits have followed each of the policies above named and we can look forward to still better records of sound teeth when simultaneous use of all three is undertaken.

As for accidents, the last of our list of twelve, we must admit that so far public efforts have had meager results, best under conditions of industrial employment, but only in exceptional circumstances on the highway and in homes. Techniques are being developed and one can take courage from the experience of Dr. Prothro at Kalamazoo, Mich., that a community can be made sensitive to the danger of home accidents and will respond helpfully to health department leadership with efforts to reduce these.

To what end have I dragged you systematically through this dozen of headings? Because in the first place there seems to me to be a tendency to divert the resources of public health departments to purposes for which they were not intended and that otherwise qualified agencies can fulfill as well or better. Because no other body of professional servants of science and the public is so well qualified to deal with preventability of disease and make it work for the public good. Because it seems to me if officers and health department staffs would concentrate on prevention they will be so well occupied and so satisfy their public that their energies will not be diverted by specious arguments into the fields of medical care of the sick.

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Diabetes Mellitus and Diabetic Retinitis

Factors Influencing Regulation

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• Fifty patients with diabetes of long duration —20 to 35 years—who followed regimens to control the disease with the greatest fidelity did not have visual complaints; retinal abnormalities were minimal and hypertension, albuminuria and renal impairment were absent.

Diabetes is a disease of total metabolism and not related solely to carbohydrate. In the pre-insulin era many facts concerned in the regulation of diabetes were established scientifically —facts such as that regulation of the body mass and control of obesity are important, that damage is caused by over-restriction of carbohydrate intake, and that hyperglycemia activates diabetes. Many failures in the treatment today are owing to insufficient attention to these basic factors. Good control requires an effort to keep hyperglycemia and glycosuria at a minimum.

NOW THAT INSULIN has been in general use for more than thirty years in the treatment of diabetes, it seems worth while to review the results of treatment and particularly the results of various types of treatment. First it might be well to define diabetes and to consider the nature of the disease. Like most textbooks, the most recent and most universally used textbook on medicine describes diabetes mellitus as "a disorder of carbohydrate metabolism characterized by hyperglycemia and glycosuria." But if only the carbohydrate metabolism were involved, treatment would be reduced to a simple equation and there would need be no further concern regarding the safety of the patient or his future. That such is not the case is reason for review of some of the other, and equally important, factors which regulate the disease.

The Naunyn era extended from 1898 to 1914. Carbohydrate intake was restricted ad infinitum, while fat and protein were forced to the limit of digestibility. To combat the effect of carbohydrate restriction, overfeeding was insisted upon, a measure which, confoundingly, brought about rapid weight loss and emaciation. Gangrene, severe retinal hemorrhages and cataracts were exceedingly common. A great deal of an attending physician's time was occupied in forestalling acidosis and combating impending coma. Coma was the universal cause of death. The average duration of life of a diabetic child was only three to six months.

In the Allen era, 1914 to 1921, the treatment of diabetes was brought from a state of empiricism to one of some scientific accuracy. In this brief era the life span of diabetic persons was more than doubled. Diabetes was shown to be a disease of total metabolism and not one solely related to carbohydrate. Too close restriction of carbohydrate was shown to be harmful. The principle upon which this newer form of treatment rested was a restriction of the total calories to the amount which the patient was capable of utilizing.

Today, no physician would recommend undernutrition in any sense of the word, but all should be guided by scientific information gained in that period. At least it was learned that overnutrition and careless diets were unwarranted. This was a difficult era. Many disconcerting stigmata were attached to it, not only because of the low calory diets, emaciation, weakness and death, but because any form of treatment was unsatisfactory. The number of physicians who practiced during that era and saw the distressing ravages of the disease is rapidly decreasing. Today the disease is masked with insulin.

Through clinical and animal experimentation certain basic facts were established. Diabetes became quiescent with reduction of the body mass and metabolism. High calory diets activated diabetes rapidly. The more liberal the diet, the more rapid the loss of weight. Following the onset of diabetes with polyuria, polydipsia and weight loss, patients were never able to retrieve any of the weight thus lost. Hyperglycemia was shown to be harmful. Well controlled diabetes became rapidly activated in the presence of hyperglycemia. The experimental production of hydrophic degeneration of the beta cells of the pancreas in the presence of hyperglycemia and the reversion to normal cells when hyperglycemia was abolished bears considerable scientific weight. One

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thing which made a firm impression in that era was the rapidity with which diabetes progressed when the metabolic load was increased by tuberculosis, infection, hyperthyroidism, obesity, acidosis or pregnancy. Diabetic women rarely became pregnant and when they did early termination was necessary to save the mother's life. The rapidity with which wounds and infectious diseases healed in animals and humans following the restoration of normoglycemia with low calory regimens was an amazing and revealing phenomenon. This was related to the restoration of normal water balance and the alleviation of chronic dehydration which accompanies hyperglycemia. Later, Bird and MacKay[6] in 1932 showed that dehydration exerted a profound influence on wound healing.

Thus, in this era, a definition was established—that diabetes is a condition in which the patient is unable to utilize the normal amount of food. Food is measured in calories. Sugar and carbohydrate are not specific factors in diabetes. The only relation which carbohydrate bears to diabetes is the law of diminishing return—the greater the concentration of the carbohydrate, the less it is utilized.

INSULIN

Within a brief period of years following the discovery of insulin, it was recognized that it was not a panacea. Its principal value was that it kept patients alive. In order to control the disease successfully it is necessary to make use of facts regarding regulation which were learned in the preceding era.

One of the first of the clinical experiments was the high fat diet of Newbergh and Marsh,[19] who recommended daily intake of 200 to 300 grams of fat and restriction of carbohydrate. With this diet glycosuria could be abolished for a short time, but diabetes soon became severe and uncontrollable—a fact which had been previously demonstrated in the Van Norden and Allen eras. Then came a period of high carbohydrate diets.[27] The diets were low in fat. Any success which this form of treatment possessed lay in the previously known fact that carbohydrate can be increased appreciably when the total caloric intake is restricted by the limitation of fat. Other forms of diet have been advocated: high protein, alcohol, inulin, odd carbon fat, and many others.

After insulin became generally distributed in 1922, physicians treating diabetic patients were soon confronted with the difficult and obstinate problem of obesity. It was soon realized that insulin was not the sole answer in diabetes and that obesity complicated management. In the author's experience those diabetic patients who have avoided obesity have shown consistently the best results. There is some relation between the insulin requirement of the body tissues and the caloric equivalent of the tissues; that is, the body fat has an insulin requirement much greater than the body protein structures. The successful treatment of diabetes is not a matter of keeping a patient aglycosuric with liberal diets and large amounts of insulin. It is possible to keep patients aglycosuric with huge quantities of insulin and high calory diets, but vascular complications occur early when that is done. Successful treatment is a matter of avoiding overnutrition and not subjecting the patient to a metabolic load over and above that required for normal nutrition.

CONSTANT NORMOGLYCEMIA

Normoglycemia, although it is not possible to maintain it throughout each 24-hour period, should be the object of treatment and control. The best results have been obtained in those patients who have most consistently avoided glycosuria and hyperglycemia. As hyperglycemia and glycosuria are nonphysiologic to say the least, it is logical to believe that patients who avoid these abnormalities for the greatest portion of each 24 hours will be freest from active diabetes. In diabetes it is not possible to maintain constant normal metabolite balance, but this is no reason to be discouraged from attempting it. Tissues which are constantly bathed in solutions of molecular concentration greater than normal may not be expected to remain normally viable over any extended period.

Caloric restriction should be such as to provide a diet adequate in calories for daily activity, but with no surplus. Protein in the diet deserves the first consideration; it should be normal for the individual. The amount of carbohydrate needed usually is from 150 to 200 grams. Caloric restriction is accomplished principally by limiting the amount of fat in accord with the nutrition, weight, duration of diabetes and state of diabetic control.

CONTROL OF HYPERGLYCEMIA

The progress of 50 diabetic patients controlled from the standpoint of hyperglycemia and glycosuria and followed 25 or more years by Reuting[21] is very heartening. Reuting's report was a follow-up of one made by Shepardson[29] in 1929 upon patients who at that time had had diabetes for five years or more and were less than 40 years of age. There was no instance of blindness in the group reported upon by Reuting even though the average duration of diabetes was 28.4 years.

In the excellent work of Jackson and associates[11]

it was observed that degenerative lesions were less prevalent in children in whom hyperglycemia and glycosuria were continuously controlled. Ricketts[22] expressed the belief that until hyperglycemia is scientifically proved to be harmless, it is unsafe and unwise not to strive for normoglycemia. White[35] in a report on a large series of juvenile patients called attention to the importance of dietary control in preventing vascular lesions. Mosenthal[18] emphasized conservation of body protein, which is destroyed in the presence of glycosuria; and he also stressed the effect of chronic dehydration associated with glycosuria as a factor in tissue injury. Jackson[12] observed that juvenile patients with well controlled diabetes tolerated exercise with less risk of reaction than did patients in whom the disease was poorly regulated. White,[36] reporting upon a study of 220 diabetic juvenile patients with diabetes of over 20 years' duration, noted that the incidence of vascular lesions depended upon the degree of control. Joslin's[13] vast statistics are convincing as to the value of treatment in reducing the incidence of vascular complications. He called attention to the work of Dolger[7] and others, and stated: "We are greatly indebted to them for bringing home to us so forcibly that many patients with diabetes of 25 years' duration show trouble with their eyes and their vascular systems. These more recent graduates have had opportunity to study your and my poorly treated cases, but ten years from now they can report success with our better methods of management." Root[26] reported upon patients in whom diabetic neuropathic changes were owing to lack of control by diet and insulin rather than to the severity of diabetes. Root and Sharkey[25] noted extraordinarily early incidence of vascular disease in obese patients. Early appearance of vascular disease was associated with lack of control in Wilson's[37] series of several hundred patients with diabetes of long-standing. Patients who had had diabetic coma once or more after diagnosis had more vascular lesions than those in whom the disease was well controlled.

Kimmelsteil-Wilson syndrome has not been noted in patients in whom hyperglycemia is consistently controlled.

THE RETINAL CHANGES IN DIABETES

Formerly retinal changes in diabetes were considered to be of arteriosclerotic origin,[33] but it is now known that that is not the case.[32] With the prolongation of life with insulin, the classical retinal picture in diabetes can be recognized in young persons who have neither arteriosclerosis nor hypertension.

As early as 1875, Leber[15] published observations on pathologic changes in the retina in diabetes. He described the round punctate spots in the perimacular area which now are well known. Undoubtedly the earliest pathological changes in diabetes occur in the retina. In 1876, MacKenzie[16] also described the ophthalmoscopically observable changes of diabetes much as they are known today. His illustrations of microaneurysms which he observed in fresh tissue are identical with those of later observers.[1] In 1943, Ballantyne[3] called attention to capillary telangiectases or aneurysms which he thought might be specific in diabetes (Figure 1). Further work by him,[5] and investigations by Friedenwald,[9] Ashton[1] and others indicated that there is rather high incidence of such lesions in uncontrolled diabetes. In a recent report by Ashton[2] it was noted that

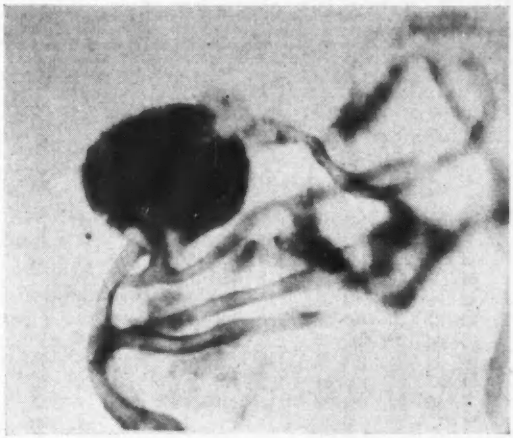

Figure 1.—"Microaneurysms in unstained retina, viewed on the flat. They are attached to capillaries" (A. J. Ballantyne and A. Lowenstein, Brit. J. Ophth., p. 594, Dec. 1944.) Also see reference 16.

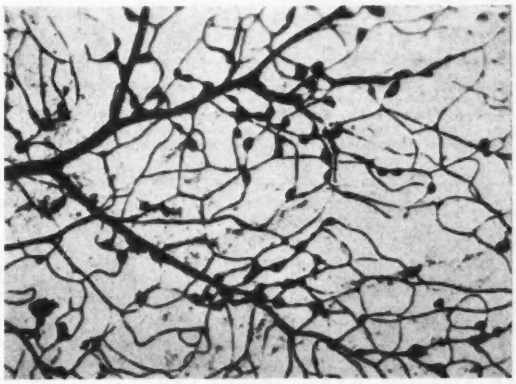

Figure 2.—Capillary microaneurysms can be found ophthalmologically in untreated diabetes as early as the twelfth to fifteenth year. They occur at a much later date in thoroughly controlled diabetes and, although visible, they need not necessarily interfere with vision (Ashton, N.: Retinal microaneurysms in the nondiabetic subject, Brit. Jr. Ophth., 35:189, 1951).

TABLE 1.—*Persons in whom diabetes developed before the age of 17 and who have had diabetes from 20 to 30 years without visual disturbances or visual complaints. All came under supervision early and at no time were subjected to a high calory dietary. The calories in the diet have been adequate for their normal work and environment. Albuminuria, renal disease and hypertension are consistently absent. Minor retinal changes are present, but vision has been preserved in spite of the added burden of pregnancy in many. There were ten successful pregnancies in seven of these patients.*

Sex	Age at Onset	Present Age	Duration, Years	Blood Pressure	Weight (Pounds) Maximum	Weight (Pounds) Present	Height	Insulin: Protamine Zinc	Insulin: Regular	Present Diet: Protein (Gm.)	Present Diet: Fat (Gm.)	Present Diet: Carbohydrate (Gm.)	Present Diet: Calories	Retinal Changes †	Condition of Eyes	Children
F	2	31	29	130/70	126	113	5′4″	18	8	....	....	.....	1700	I	Vision 20/20.	....
F	14	44	30	140/90	117	110	5′1″	24	20	....	....	.....	1600	O	Vision 20/20.*	3
F	2	32	30	135/82	115	109	5′3″	30	10	71	74	185	1685	I	One small scar.	....
F	2	32	30	140/85	130	126	5′4″	44	20	85	70	170	1650	I	One small hemorrhage.*	....
M	15	44	29	135/80	225	200	6′0″	50	30	....	....	.....	1705	I	20/20 vision.*	....
F	10	34	24	124/84	138	125	5′5″	26	20	90	90	150	1770	II	Punctate hemorrhages, aneurysms.*	1
F	8	33	25	120/81	137	126	5′2″	35	25	....	....	.....	1500	I	Vision 20/20.	1
F	8	30	22	120/72	116	97	5′2″	18	9	72	72	140	1596	I	Few aneurysms. Vision 20/20.*	1
F	14	38	24	140/100	134	133	5′6″	22	18	90	60	165	1560	I	Two aneurysms. Vision 20/20.	1
F	7	31	24	132/86	155	154	5′2″	45	22	90	60	165	1560	I	Vision 20/30. No hemorrhages.*	....
F	8	28	20	130/86	153	140	5′4″	18	12	90	50	155	1430	I	Vision 20/20. Few punctate aneurysms.*	....
F	16	42	26	140/90	125	125	5′8″	38	12	80	73	182	1705	O	Vision 20/20.*	2
F	3	23	20	121/81	133	114	5′5″	24	10	85	70	170	1650	II	Astigmatic, aneurysms, two exudates.*	....
F	10	32	22	100/80	147	135	5′3″	41	30	100	80	150	1720	I	Vision 20/20. Few aneurysms.	1

*Retinal examination and report by members of American Board of Ophthalmology.
† The extent of severity of retinal changes is on a grade scale of zero to IV.

TABLE 2.—*Patients in whom diabetes developed after the age of 18 and who have had diabetes from 20 to 38 years without visual disturbance, although there are minor retinal changes. These patients consistently followed a low calory regimen and they prevented glycosuria and hyperglycemia as constantly as possible. They also avoided obesity. Symptomless hypertension was noted in three persons. Albuminuria was inconsequential when present. There was no evidence of renal disease. This table illustrates the point that visual disturbances can be retarded, although minor grades of retinal changes such as aneurysms, small punctate hemorrhages, etc., may be present for many years.*

Sex	Age at Onset	Present Age	Duration, Years	Blood Pressure	Weight (Pounds) Maximum	Weight (Pounds) Present	Height	Insulin: Protamine Zinc	Insulin: Regular	Present Diet: Protein (Gm.)	Present Diet: Fat (Gm.)	Present Diet: Carbohydrate (Gm.)	Present Diet: Calories	Retinal Changes †	Condition of Eyes
F	39	68	29	170/100	175	144	5′6″	25	....	84	60	175	1580	I	Minor aneurysms.
F	25	49	24	137/100	148	148	5′6″	35	20	90	75	190	1815	I	Few aneurysms.*
M	36	71	35	110/79	157	157	5′11″	40	25	90	80	190	1840	I	Vision 20/20. No aneurysms.*
F	30	67	37	190/90	200	165	5′2″	25	20	85	50	160	1430	II	Several small hemorrhages and exudates.
F	22	43	21	118/72	122	118	5′3″	28	13	90	80	180	1800	II	Few aneurysms and exudates. Vision 20/20.*
M	41	72	31	137/81	230	160	5′9″	35	24	100	75	180	1795	I	Few aneurysms. No exudates.*
M	35	57	22	140/80	204	165	5′8″	27	20	110	75	190	1875	O	No hemorrhages. No exudates.*
F	20	43	23	200/100	106	95	5′0″	22	11	72	72	135	1424	I	Aneurysms and exudates.
F	54	80	26	167/80	180	160	5′6″	24	14	84	60	175	1580	I	Vision 20/25.
F	24	54	29	180/95	166	147	5′5″	30	15	80	38	150	1262	O	No hemorrhages.*
F	26	50	24	192/90	159	127	5′3″	22	14	81	45	120	1209	I	Vision 20/25. Punctate hemorrhages.
F	36	59	23	216/110	187	182	5′8″	32	18	85	50	160	1430	I	Myopic. Macular aneurysms.*
M	28	66	38	160/95	157	135	5′8″	25	14	80	80	160	1680	I	Minor macular aneurysms. No hemorrhages.*
F	26	51	25	127/80	148	148	5′8″	18	15	84	60	175	1580	I	Few aneurysms, minor exudates. Vision 20/20.*
M	20	56	36	143/82	175	165	5′10″	25	20	90	75	190	1815	I	1 or 2 aneurysms. Vision 20/20.*
F	34	56	22	159/79	157	152	5′5″	36	17	80	70	150	1550	II	Few aneurysms. Few exudates.*
F	22	53	31	141/85	153	147	5′10″	38	24	90	75	180	1755	I	Congenital cataract left. Right retina normal.
M	18	41	23	133/70	150	143	5′10″	30	20	100	100	195	2175	I	Vision 20/20. Aneurysms.
M	29	57	28	140/88	170	136	6′0″	32	20	100	120	190	2240	II	Aneurysms. Fresh hemorrhages.
M	45	68	23	127/70	130	96	5′0″	35	25	....	....	.....	.....	I	Aneurysms. Vision 20/25.
M	29	56	27	120/79	190	175	6′0″	40	35	100	100	210	2180	I	Old scars, microaneurysms.
M	47	68	21	134/86	230	152	5′7″	0	0	100	75	120	1555	I	Vision 20/20. Microaneurysms only.
F	19	41	22	119/70	132	126	5′9″	36	....	75	75	150	1575	O	No hemorrhages or aneurysms.*
F	51	72	21	138/86	135	119	5′1″	30	20	75	60	150	1530	O	No hemorrhages or aneurysms.*
M	29	55	26	120/79	175	165	5′11″	40	30	100	100	200	2140	II	Microaneurysms and fresh hemorrhages.
F	48	70	22	130/80	148	127	5′6″	20	15	75	70	150	1535	II	Perimacular aneurysms.

*Retinal examination and report by members of American Board of Ophthalmology.
† The extent of severity of retinal changes is on a grade scale of zero to IV.

retinal microaneurysms occur in nondiabetic subjects in association with practically all types of clinical disorders (Figure 2). Microaneurysms, associated with glaucoma in most instances, were observed in 30 of 89 eyes removed at operation. In postmortem examination of 85 eyes of subjects who had had nondiabetic diseases of all types, aneurysms were observed in 29.

As seen with the ophthalmoscope the lesions are round and discrete with very clear outlines and are located in the macular and perimacular areas. They are not associated with the large vessels. Further studies have shown the lesions to be microaneurysms[4, 1] with endothelial walls. With specialized staining technique and serial sections, afferent and efferent vessels may be clearly outlined (Figure 3). These microaneurysms are located in the inner nuclear layer, and as the overlying structures are elevated, the lesion appears to be spherical or globoid. They vary from 30 to 60 microns in diameter—just within the limits of visual acuity. Light reflex may be noted in many of them. In fresh or stained specimens great numbers can be seen that are not visible on fundoscopic examination. As the lesions progress, erythrocytes may escape through the endothelial aneurysmal wall by diapedesis; also the aneurysm may rupture and definite hemorrhage occur. Fluctuation in intraocular pressure is known to accompany variations in the content of sugar in the blood.[14] Both extracellular and intracellular fluid balance is altered when the content of metabolites in the blood departs appreciably from normal.[8, 28] In uncontrolled diabetes such fluctuations in retinal capillary pressure would invite aneurysmal hemorrhage owing to interference with the functional integrity of the vascular endothelial lining. Ashton[1] confirmed Ballantyne's report that the globular lesions of the retinal vessels are true capillary microaneurysms.

Curiously, the lesions are confined to the eye.[1] McCulloch[17] reported capillary aneurysms in the conjunctivae in diabetes. Aneurysmal dilations have not been observed in examination of various other tissues from diabetic persons — lungs, meninges, liver, pancreas, brain, pleura, peritoneum, etc. Diabetic aneurysms are round and uniform in size in contradistinction to the elongated and irregular hemorrhage of hypertensive and renal disease, the lesions of which are found in the middle layers of the retina.

If the aneurysms are watched closely, it may be noted that they disappear when the disease is brought under control. This was illustrated in Case 8 (Table 1) of the present series, during the course of pregnancy in a woman 21 years of age with diabetes of 13 years' duration. With the added strain of advancing pregnancy myriads of round discrete aneurysms were noted in the perimacular area of both eyes. Pregnancy was terminated at the eighth month. Diabetes came under control very quickly and within a month only a few red dots could be seen. This probably represented a rapid advance in retinopathic changes associated with a rapid increase in the metabolic load, a factor which is well known in the relation of pregnancy to diabetes. The author has also observed other instances of disappearance of aneurysms when good control was restored. O'Brien and Allen[20] reported that retinopathic changes disappeared after two months of good control in six diabetic persons, all of whom were under 31 years of age.

Most observers report the appearance of the lesions as occurring about the tenth to fifteenth year of diabetes. The time of onset depends upon the degree of dietary control. In Tables 1 and 2 there are instances of diabetes of 25 to 30 years' duration without the appearance of aneurysms. The author can only attribute this to the fact that the patients had adhered to a low calory regimen as closely as possible over the years. The lesions occur much earlier, sometimes within five years, in patients who have flagrantly disregarded diabetic management. Diabetes affects the veins rather than the arteries. The central veins sometimes are very large.

PRESENT SERIES

Some of the patients in the present series have been under the author's supervision for more than 30 years. They were selected for inclusion in the group here reported upon because of long duration of diabetes and their outstanding fidelity, patience and perseverance in adhering to dietary programs, the essence of which is restriction of total calories. They were also selected because most of them came under the author's supervision early in the disease—in some instances on the day of diagnosis—and had never been subjected to high fat or high caloric regimens for a long period.

None of the patients had complaints regarding vision at the time this report was written, and many had normal visual acuity. None had cataracts. In critical examination of the fundus of the eyes, only minor retinal lesions were observed and in some instances there were none. To the patient, good vision is of more concern than minor alterations in the retina. In all the patients the nitrogen content of the blood was normal. Albuminuria was present in only a few instances, and in those it was inconstant and incidental. No abnormalities were noted on microscopic examination of the urine. The blood pressure of all patients was essentially normal (Tables 1 and 2).

Table 1 gives data on patients in whom diabetes developed before they were 18 years of age, Table 2 on those in whom the onset of disease occurred at a later age. Those in the former group escaped not only retinitis but the other hazards related to advancing years, such as coronary occlusion, hypertension and peripheral vascular sclerosis. The latter group escaped renal disease. None of the patients experienced hardship in avoiding the hazard of obesity in diabetes. The patients in the older group were willing to reduce body weight, and those in the younger group avoided obesity. All the patients in the younger group (Table 1) have been under the author's supervision for 20 to 30 years, and at no time had they been on high calory diets. The young female patients have maintained normal vision despite the added burden of pregnancy, in some instances multiple pregnancies.

DISCUSSION

It is an accepted principle in medicine that any process which deviates from normal is not only pathological but harmful. There is abundant evidence that hyperglycemia is harmful, and none at all, as Ricketts[23, 24] has observed, that it is harmless. There is sufficient evidence that it induces chronic dehydration which is a major factor in tissue injury.[6] Weight gain of two or three pounds within a period of 12 to 24 hours after restoration of normoglycemia in a person who previously has had uncontrolled diabetes is a matter of everyday clinical experience. Hyperglycemia interferes with the normal osmotic processes in the normal cells.[31] It is reasonable to assume that body tissues which are constantly bathed in fluids of abnormal chemical composition will undergo pathological change. The development of cataracts in frogs that are confined in hypertonic glucose solution[10] is an academic example. As long ago as 1860, Mitchell[17a] showed that some accompanying degree of desiccation was necessary in addition to hyperglycemia in order to produce cataracts in frogs. In either event, hyperglycemia is harmful either per se or because of the inevitable dehydration. The fact that hyperglycemia produces degenerative changes in the beta cells of the islands of Langerhans and that these changes are reversible upon restoration of normoglycemia must be considered. Arrest in development of potential diabetes can be accomplished by control of hyperglycemia, as was shown by Watson.[34] By the same token it is logical to assume that progression of actual diabetes can be arrested. This was very well illustrated by Sherrill[30] in a report on a series of extremely severe cases. Patients soon appreciate the freedom from thirst, polyuria, neuritis, cramps in the legs, blurred vision, pruritus, fatigue, weakness and loss of weight that comes about after hyperglycemia is controlled.

Since ocular aneurysms have been observed in pathologic examination of specimens taken from nondiabetic subjects (although they are not seen on ordinary ophthalmologic examination) question arises as to why the aneurysms in persons with uncontrolled diabetes progress to serious proportions whereas ordinarily they cause little trouble when associated with other diseases. It must be assumed that uncontrolled diabetes is the deciding factor, and therefore that hyperglycemia is harmful. In uncontrolled diabetes there is also a disturbance in the nitrogen balance, which brings about the dearth of protein in the plasma that occurs in patients with untreated diabetes of long standing. The relationship of low content of protein in the plasma to retinal hemorrhages is known, and it has been shown that vision and the retinal condition can be improved with adequate treatment and high protein feeding.

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Gamma Globulin Distribution

THE OFFICE OF DEFENSE MOBILIZATION has taken over responsibility for distribution of gamma globulin for use in treatment of paralytic poliomyelitis, infectious hepatitis and measles. Details of distribution are being evolved by a National Research Council committee of physicians. It is understood the plan contemplates distribution of the central supply of gamma globulin to state health officers, who will be responsible for its final local use.—A.M.A. *Washington Letter*.

Mental Medication for Your "Sick" Patient

JOHN B. LONG, M.D., Sacramento

• *Essential stages in a type of superficial psychotherapy that has been found to be generally effective in treating a patient with "normal" neurosis are: A complete medical health evaluation, achievement of the patient's confidence, a description of all findings and a convincing explanation of how certain symptoms may be caused by nervous tension, and appropriate medical treatment combined with encouragement, inspiration and common-sense advice.*

The "sick" patient should be convinced of the true cause of the symptoms that distress him, taught to recognize the neurotic basis of symptoms, and at times to accept and live with them. The fundamental ingredient of psychotherapy of this type is emphasis on developing a confident and purposeful way of life. Each patient should be encouraged to help himself in a determined effort to acquire peace and harmony both within himself and with his environment. Differences in personality and environment vary the treatment and advice best suited to each patient.

A proper attitude by the physician is a major factor in the effectiveness of treatment.

WE LIVE IN A civilization of mental and moral confusion, and *we are all neurotic.* The very awareness that one is not as perfect as he would like to be produces distress. At various times every intelligent person has some form of neurosis, with acute exacerbations and remissions. A neurotic person is anyone who experiences physical symptoms from emotional stress.

In every person there exists a Dr. Jekyll and a Mr. Hyde living within the same brain and engaged in a continual struggle for expression. The resultant conscious and subconscious strain is expressed not only as emotional tension but also as physical illness. Each person, in addition to the struggle within himself, is engaged in a persistent struggle with his environment in order to gain the necessities of life. The restrictions of society create many difficulties in our fight to gain personal significance, self-confidence, prestige, sexual satisfaction, and other normal human cravings. Whenever environmental stress becomes too great, or when mental conflicts become overwhelming, neurotic symptoms follow. In most cases the symptoms result from various combinations of both external and internal struggles. Life is synonymous with conflict, from the cradle to the grave. This concept of the *normalness of a neurosis* has helped the author to see a way through the confusing maze of symptoms presented by the average patient.

THE ROLE OF THE GENERAL PHYSICIAN

Since the diagnosis of mental or emotional illness is frequently considered an insult by patients or by their relatives, the attending physician is faced by two alternate modes of practice: that of attributing all of the symptoms to the minor physical abnormalities observed, or that of taking the time and effort, at the risk of offending, to adequately explain how each symptom may express a combination of physical and emotional factors. The latter course, which entails convincing a person who does not want to be convinced, requires art, skill and patience. It is the hard course of medical honesty.

All that may be needed for most patients is a bit of reassurance and friendly advice; often calming a fear may help the patient to weather an emotional storm. The explanation of how distressing physical symptoms can be produced by distressing thoughts frequently brings about the return of sufficient happiness and health to make further medical care unnecessary for long periods.

Other patients may need repeated medical attention and reassurance. How convenient it would be to send these troublesome and time-consuming patients to a psychiatrist and let him worry about them. But physicians in general practice have learned that, for several reasons, this is seldom possible. Often it is necessary to continue to care for patients who may remain almost continuously fearful, fatigued, complaining and ill. It is easy to become discouraged in attending these intrinsically neurotic and often incurable persons unless it be borne in mind that many organic conditions also can be only partially alleviated and not cured. It is helpful to remember that repeated reassurance given

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to a chronically "sick" person is an essential part of treatment.

The great bulk of functional disease, both acute and chronic, has been by necessity managed by personal physicians rather than by psychiatrists. Although this type of care may not always be the best for the most severe constitutional neurosis, still the general practitioner is in a better position than the psychiatrist to treat ordinary neurosis. One obvious reason is that the most prominent symptoms of emotional tension are those of physical dysfunction, which the patient interprets as owing to organic disease. The patient goes to his attending physician for relief of headache, palpitation, fatigue or indigestion. The confidence the patient places in his own doctor to diagnose and treat his physical sickness is of great value in effective psychotherapy. Reassurance that the distressing physical symptoms are the normal bodily reactions to emotional tension can best be accepted from the one who has just conducted a complete health evaluation.

Physicians in general practice, each in his own way, attempt to treat both the organic and functional ills of their patients. In no phase of the practice of medicine, however, is there a wider range of equally effective methods than in the treatment of functional illness. Probably no two physicians treat neuroses in the same way, but each has developed a treatment method which, for him, is effective.

The author, in discussing his own way of treating patients with a neurosis, will not attempt to present scientific facts but only ideas, concepts, opinions and practical suggestions that have been gathered from various persons, lectures and books.

STEPS IN TREATMENT

Obviously, the first and most important step in psychotherapy for the "sick" patient is a complete medical examination. This includes whatever laboratory and x-ray studies may be needed to verify the clinical diagnosis and convince the patient that the physician is giving due consideration to physical complaints.

The second essential is to convince the troubled patient that he is being treated by a capable physician who understands the patient's illness, and knows what to do about it. The patient must be convinced that the physician likes him as a person and can show him the way to physical and emotional health if he is willing to follow.

The third step is a description of all physical, laboratory, x-ray and emotional findings, and next how they are related to each symptom. It is essential that the patient be made to understand that nervous tension is average and normal, and that it occurs almost exclusively in intelligent, sensitive, kindly and essentially good people. Explanation of how the symptoms can be produced by nervous tension must be both convincing and acceptable.

The fourth step is the administration of appropriate medical treatment mixed with sufficient common-sense advice and encouragement to bring about symptomatic relief. With the relief of distressing symptoms the patient gains new faith in the physician, in himself and in the world about him. With faith comes a willingness to accept a diagnosis of functional illness and a desire to develop a more wholesome "way of life."

Usually three or four visits are required for the average neurotic patient. On the first visit, after the physical examination has been completed, the purely organic conditions are described and appropriate treatment is outlined. Next comes an attempt to convince the patient first that his symptoms are well understood, and second that these symptoms can develop from nervous tensions. The patient is then encouraged to discuss his problems and disturbing thoughts. After the patient has finished talking, a few words of sympathy and reassurance are followed by a brief discussion of whatever environmental manipulation might obviously be helpful. One should be careful not to tell the patient what he should do, but, by asking leading questions, to help him reach a solution himself. The visit is usually concluded with an optimistic assurance that the symptoms will disappear as the emotional problems are corrected. Usually sedative medication is prescribed to speed relaxation and symptomatic relief.

On the second visit a few portions of the physical examination are rechecked. The laboratory and x-ray findings are explained. The final diagnosis is then given—again listing organic conditions first. The patient is encouraged to talk further about his problems and what he intends to do about them. Some reference may be made to the frequency with which poor sexual satisfaction is associated with emotional problems. This approach may open the door to a discussion of a major factor in the production and continuation of tension. No attempt is made to delve deeply into the guilt complexes or disturbing psychosexual aberrations of early life. In fact all discussions with the patient are conducted with a reassuring emphasis upon helping him, a normal person, to face life with a better understanding and a greater peace of mind. The patient is encouraged to change things that should be changed, but to accept situations that cannot be changed. It is helpful to advise the patient to try to live one day at a time to the best of his ability, giving no thought to the mistakes of yesterday or to the insecurities of tomorrow. The second visit is often concluded by a gift to the patient of the little book *A Way of Life* by Sir William Osler.

Further details of therapy cannot be standardized. Various personality patterns, differing religious beliefs, and specific environmental factors determine what type of subsequent therapy would be best suited to each patient. An understanding of how environmental trauma can injure a sensitive personality sufficiently to produce physical illness will in most cases reveal to the patient what must be done to remedy the situation. One patient may decide to change some phase of his environment, another may change himself, and still another may attempt to do both. One patient may find peace of mind by accepting the inevitable, another by accepting the sustaining comfort of religious faith. All patients are benefited to some degree by kindly guidance in their search for happiness and emotional security. With the aid of a practical philosophy they can be helped to regain self-esteem and self-confidence. Personal significance is established by focusing their attention upon a recognition of their worthiness and upon their capability to bring happiness to others. They are urged to look for the beauty in life and for the good in others. They are assisted in their attempts to balance the ever-present hates and resentments with tolerance. They may be cautioned not to expect perfection in themselves or in those with whom they live and work. They are reminded that life does not offer security, only opportunity. They are urged to concentrate upon *something to live for* if they would make the battle of life more endurable. They are encouraged to develop *enthusiasm for a cause,* for *earning happiness* by giving happiness. They are persuaded to face each day with a determination to concentrate their efforts and attention upon doing, one at a time, those things that can be done—to crowd disappointments and uncertainties out of their minds by substituting fruitful activity.

AVOIDING DEEP-LYING CONFLICTS

Admittedly this is a very superficial form of psychotherapy, but that is intended. It must always be kept in mind that it may not be wise for physicians who are not psychiatrists to probe too deeply into the repressed complexes or psychic irregularities of all patients. Neurosis itself may be an essential and necessary part of some personalities, and to remove the physical symptoms caused by it might expose the patient to an even more excruciating mental pain. A physician might bring great harm to some patients by emphasizing their abnormalities, by discovering repressed sexual deviations, or by exposing subconscious shame-producing urges. Certain danger signals that indicate a major mental disturbance must be recognized, and when they are observed the patient would best be referred immediately to a psychiatrist or placed in protective custody. Psychiatric symptoms of that order are: hallucinations, delusions, emotional extremes with suicidal or violent tendencies, obsessive compulsive rituals, depressions with withdrawal symptoms and excessive feelings of unworthiness.

THE PHYSICIAN'S ATTITUDE

It is admittedly difficult for a busy physician adequately to treat the multitude of functionally ill persons. He may find it relatively easy to devote the necessary time to patients whom he spontaneously likes, but the greatest challenge in the general practice of medicine is to deal properly with patients who are unpleasant and unhappy, irritable and antagonistic, whining and complaining, depressed and discouraged.

To help them, a physician must gain their trust and faith. And to do that, the physician himself must be emotionally oriented to the task. He must first of all believe that it is worth while, and he must have honest sympathy for the patient's suffering, whether the cause be physical or emotional. He must believe that a degree of self-respect and peace of mind is essential to both mental and physical health. In short, he must have a conviction before he can convince. If he can develop this conviction he will be a more effective therapist. From a more selfish point of view, any attitude that improves a physician's relations with difficult-to-handle patients should make his work easier and happier. Some concepts and rules that have been helpful in this matter are:

1. *Appeal to the natural desire of each patient for approval and respectability.* Don't resort to flattery, but look for qualities that you can genuinely admire. There is both good and bad in every man; speak to the good and the good will answer. This is especially true of those who may be antagonistic and emotionally insecure. Talk up to each patient, not down to him.

2. *Make a habit of attempting to place yourself in the patient's position.* View the symptoms and problems through his eyes. Never suggest a course of action which the patient cannot or would not be likely to follow, even though such action would appear to be logical.

3. *Think of each neurosis as normal.* Assume that the neurotic patient is an average and intelligent person who is troubled by a combination of problems which is beyond his understanding and control. View and speak of his symptoms as normal manifestations of environmental injury to a sensitive personality.

4. *Consider all illness as a combination of both organic and emotional factors.* Don't think in terms

of whether the cause is organic or functional, but rather of what combination of these two factors produces the symptoms.

5. *Keep bolstering the self-esteem and self-respect of each patient.* Refrain from ridiculing or minimizing the importance of his complaints. Present the diagnosis and treatment in such a way that it can be accepted without humiliation to the patient.

6. *Remember that what you say can produce illness, as well as cure.* Talk and act confidently about what you do know, but never share with your patient your doubts and uncertainties. Don't tell the patient that he has no physical disease and then cause doubts in his mind by ordering more tests or making repeated appointments.

7. *Keep in mind that most emotional wounds will heal with time,* but the timely application of helpful advice can speed recovery and prevent disabling scars on the patient's personality.

Despite honest effort to follow all these ideas in the treatment of an emotionally upset patient, a physician will occasionally attend a patient toward whom he feels antagonism. When this occurs, the physician should recognize his own resentment and refer the patient to a colleague who probably will not have to struggle with a similar antagonistic feeling. As a general rule, however, the treatment of a neurotic patient can actually be a pleasure for the physician if he develops the proper frame of mind.

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Army Reviewing Physical Records of 1,000 Priority-3 Physicians

THE ARMY SURGEON GENERAL has asked area surgeons in the U. S. to review records of about 1,000 priority-3 physicians who were deferred for physical reasons prior to lowering of physical standards. A number of doctors in this group are expected to be found fit for duty. Under lowered standards announced last month, each case is decided on its own merits. Generally, the armed services are taking the position that if a doctor is physically able to carry on a private practice then he is fit for military service.

Since the Defense Department announcement on lowered standards, the Army has sent to Selective Service for reevaluation the names of 739 deferred physicians in priorities 1 and 2. Of this group, 487 have received physical examinations.—A.M.A. *Washington Letter.*

Lymphoblastomas in Childhood

Cutaneous Manifestations

LAWRENCE M. NELSON, M.D., *Santa Barbara*

• *The lymphoblastomas occurring in childhood are divided for purposes of discussion into lymphocytoma cutis, mycosis fungoides, lymphosarcoma, Hodgkin's disease, and leukemia. The cutaneous lesions may be either specific (as a result of the infiltration of the skin with specific cells of the conditions) or toxic (nonspecific). With the possible exception of mycosis fungoides, the cutaneous manifestations are not diagnostic. The final diagnosis depends upon microscopic examination of the specific tissue involved and the coordination of the clinical and microscopic findings.*

THE TERM *lymphoblastoma*, while open to certain legitimate criticism, has become fixed in medical literature and, to dermatologists at least, has a very definite meaning. While not all dermatologists agree as to just what conditions should be classed as lymphoblastomas,[1] most of them consider mycosis fungoides, leukemia, lymphosarcoma, and Hodgkin's disease to be the more important members of the group.[4, 52] Among the less important and even debatable members are lymphocytoma cutis and Spiegler-Fendt sarcoid. It is generally agreed that Spiegler-Fendt sarcoid should be classed as a lymphoblastoma.[17, 39] Spiegler's[63] patients with this disease died, as did Sweitzer's,[66] and one of Lewis's.[40] However, Fendt[21] did not give so bad a prognosis, and some of his patients either responded to therapy or had spontaneous resolution of the lesions. Bafverstedt[2] recently reclassified as lymphocytoma cutis many cases originally considered to be Spiegler-Fendt sarcoid. Lewis,[41] in discussing the paper of Loveman and Fliegelman,[43] stated that "from the evidence given by the authors, it would seem that the disease under discussion is identical with the localized form of Spiegler-Fendt sarcoid." While some observers feel that lymphocytoma cutis is always benign,[17, 43, 49] others do not share that view,[41, 48, 65a] and all agree that prolonged and careful observation is necessary to make sure there is not malignant change. Even though the question of the exact relationship of these two conditions to each other and to the other lymphoblastomas has not been settled, the author looks upon lymphocytoma cutis as a relatively benign lymphoblastoma. The condition must be considered in the differential diagnosis of the lymphoblastomas, and while it apparently remains benign in most cases, it may become malignant. In the present discussion Spiegler-Fendt sarcoid and giant follicular lymphoma will be placed in the lymphosarcoma category.[17, 31, 54, 64]

Since, to the best of the author's knowledge, no report of reticulum cell sarcoma in childhood (other than in bone[31]) has appeared in the literature, any discussion of its place in the classification will be omitted.

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Of the leukemias, the acute form is the most common in children. Chronic myeloid leukemia is uncommon, and chronic lymphatic leukemia does not occur.[12, 13]

Lymphocytoma cutis, mycosis fungoides, lymphosarcoma (including Spiegler-Fendt sarcoid and giant follicular lymphoma), Hodgkin's disease and leukemia have been reported in preadolescent children. Because the statistics on the lymphoblastomas are for the most part given in relation to decades of life, lymphoblastoma occurring in the first two decades (rather than before puberty only) will be considered in this presentation.

Lymphocytoma cutis (benign lymphadenoid granuloma of the skin, miliary lymphocytoma, lymphadenosis benigna cutis) is rare in childhood. The youngest patient of record, a four-year-old child observed by Kaufman-Wolf,[32] had lesions on the scrotum. Localized lesions are the form usually seen in children.[2] Loveman and Fliegelman[43] and Mopper and Rogin[49] have reviewed the literature and have reported cases in adults. The clinical appearance varies considerably, and the lesions may resemble epithelioma adenoides cysticum, rhinophyma, moles or basal cell epithelioma. Rarely if ever do the lesions ulcerate. Becker[5] stated that when pressure is applied to a glass slide on the skin, an apple jelly color persists, as in lupus vulgaris. Long observation and repeated studies of the blood and bone marrow are necessary to rule out malignant lymphoblastoma.

Mycosis fungoides (granuloma fungoides) is es-

sentially a disease of adults, although occasionally it occurs during the first two decades of life.[29, 42, 52, 56, 59] Apparently the youngest patient was a three-year-old boy reported upon by Hathaway.[29] He had a single lesion on the face, which responded to x-ray therapy. Ormsby and Montgomery[52] cited a report of mycosis fungoides in a child six years of age.

There is nothing in the literature leading the author to believe that mycosis fungoides in children differs in any way from that in adults. Mycosis fungoides usually begins as plaques of a rather nondescript scaling, pruritic dermatitis (the premycotic stage). The plaques become infiltrated, and tumors, which eventually ulcerate, develop. The condition spreads, and ultimately causes death. Although mycosis fungoides may remain localized to the skin throughout its course, not infrequently it becomes a generalized systemic disease with features like those of leukemia or one of the other types of lymphoblastoma.[36, 38, 46, 47, 52, 73] In the premycotic stage, diagnosis may be difficult or impossible. By the time infiltration and tumors appear, the syndrome is quite characteristic. Pruritus may be severe in all stages of mycosis fungoides. Occasionally pruritus alone is the initial symptom. Mycosis fungoides may also begin as poikiloderma atrophicans vasculare or as a poikiloderma-like eruption.[51, 69] It may resemble psoriasis, or it may begin as tumors with ulceration (the d'emblee type). Occasionally it begins as erythrodermia.[46, 59]

Leukemia, Hodgkin's disease, and lymphosarcoma have many cutaneous symptoms in common and for the purposes of this presentation can be considered together. First, however, it might be well to discuss the two special varieties of lymphosarcoma, Spiegler-Fendt sarcoid and follicular lymphoblastoma (follicular lymphoma, follicular type of malignant lymphoma, giant follicular lymphadenopathy, giant lymph follicular hyperplasia of lymph nodes and spleen, Brill-Symmer's disease), because each of them has a cutaneous picture quite different from that seen in typical lymphosarcoma. Both conditions are rare in childhood.

Spiegler-Fendt sarcoid appears in either a localized or disseminated form. Both forms occur in children.[40] The cutaneous manifestations of the localized form are apparently identical with those described under lymphocytoma cutis. In the disseminated form, generalized cutaneous or subcutaneous nodules, up to 2.5 or 3.0 cm. in diameter, develop. Plaques sometimes appear. The color of the skin varies from normal to deep crimson or deep red. The nodules grow to a certain size and then are static. Spontaneous regression occasionally occurs.

Follicular lymphoblastoma has been thoroughly reviewed by Gall and co-workers[24] and Combes and Bluefarb.[10] Gall, in a review of 69 cases, noted that in 3.5 per cent of them the onset was before the patient was 20 years of age. Eighty-nine per cent of the patients had peripheral lymph nodes, and 5 per cent of them had cutaneous involvement which consisted of "a few isolated, raised, firm, brownish to reddish nodules." It was reported that no diffused lesions were observed. Herpes zoster occurred in three cases. Combes and Bluefarb reviewed reports of 72 cases in the literature, and reported on 15 patients they had observed, 14 of them males. The youngest patient in the series of 87 cases reviewed was 15 years of age, the eldest 80. In the 15 cases Combes and Bluefarb had observed, the skin conditions resembled those of allergic eczema (including exfoliative dermatitis), chronic discoid and lichenoid dermatitis, and a third condition that was clinically diagnosed as mycosis fungoides. One patient, aged 17, had dermatitis resembling chronic discoid and lichenoid dermatitis, associated with giant follicular lymphadenopathy of two years' duration. Microscopic examination of the skin was carried out and the disease could be classified only as "chronic dermatitis."

The cutaneous symptoms of typical leukemia, Hodgkin's disease and lymphosarcoma are usually divided into the toxic or nonspecific lesions, and specific lesions—those containing true tumor cells. This division is not absolute, for so-called toxic lesions may contain tumor cells.[25, 39] In addition toxic lesions may later be the site of development of specific lesions, and sometimes tumor cells may be present in skin which, to clinical observation, seems normal.[22, 35] Specific lesions may develop at the sites of skin lesions in no way related to the lymphoblastoma (varicella,[22] trauma[35]). The nonspecific lesions are in no way characteristic, but may be suggestive of the underlying condition. Almost every type of lesion the skin is capable of producing may occur. Pruritus, either with or without skin lesions, is common. Pigmentation, macules, papules, lichenification and exfoliative dermatitis may occur,[25] and also bullous lesions,[17, 60] pustules, and vesicles.[75] Alopecia, atrophy, dryness and hyperkeratosis have been reported. Icterus, urticaria and bouts of unexplained hyperhidrosis may develop.[9] Herpes zoster may complicate almost any of the lymphoblastomas.[8, 72]

Especially in leukemia, hemorrhage in the skin and mucous membrane, stomatitis, noma, pallor and enlargement of the abdomen are not infrequent.[18] Some symptoms, such as dyspnea, cough, intestinal obstruction, abdominal pain, nausea, vomiting and diarrhea, may be owing to the clinical location of

the tumor mass, and are not characteristic of lymphoblastoma.[11, 15, 44, 53] Anemia and fever occur some time during the course of the disease in most patients. The so-called Pel-Ebstein fever is said to be quite characteristic of Hodgkin's disease, but may occur in other conditions also.

The cytologic structure in nonspecific cutaneous lesions is, of course, not characteristic and unless by good fortune specific cells are observed in the section, histologic examination can be of little help. However, as the underlying condition is usually pronounced, histologic identification may not be necessary for diagnosis.

Specific cutaneous lesions may consist of generalized exfoliative dermatitis (which may also occur as a nonspecific symptom), cutaneous or subcutaneous nodules, as well as tumors and plaques, and enlarged lymph nodes. Ulceration occasionally occurs, particularly in Hodgkin's disease.[60, 67, 73]

A special word should be said about chloroma, which is a manifestation of acute leukemia.[17] Over 50 per cent of the patients with these greenish tumors are under 20 years of age. The tumors may be cutaneous or may be located in positions to cause symptoms owing to pressure on adjacent organs.

The final diagnosis in any of these conditions depends upon the examination of specific tissue, be it skin, lymph node, bone marrow or blood, and the coordination of the clinical and microscopic observations.

Prognosis. The course of all the lymphoblastomas seems to be somewhat more rapid in children than it is in adults.[14, 62] However, there is great variation from case to case. Leukemia, in particular, usually runs an acute fulminating course. Follicular lymphoma and Spiegler-Fendt sarcoid progress much less swiftly than does typical lymphosarcoma. Lymphocytoma is usually benign.

Treatment. Lymphocytoma is very radiosensitive and, while it responds to radiation therapy, recurrence is the rule.[43, 49] It is also said to respond to arsenic. Excision may be curative.[65b] Occasional spontaneous remission occurs in this condition, as it does at times in other types of the lymphoblastomas.

Bierman[6] has given a very complete review of the treatment of the other lymphoblastomas. Follicular lymphomas frequently respond to x-ray therapy or to surgical excision. Single focus lymphomas may be removed surgically, and that treatment followed with intensive x-ray therapy (until further evaluation proves the latter to be of no additional value). In acute leukemia of childhood, radiation therapy, urethane and nitrogen mustard are of little or no value.[54] The most satisfactory treatment seems to be administration of the folic acid antagonists.[16, 19, 20, 30, 54, 70] Leukemia in adults does not respond to folic acid antagonists as well as does the disease in children.[16,19, 30] Toxic symptoms are common, and at best the treatment is only palliative. Radioactive substances are still in the experimental stage. Nitrogen mustard is palliative in Hodgkin's disease, lymphosarcoma and mycosis fungoides. It is also somewhat effective in giant follicular lymphoma. Its use is frequently complicated by granulopenia. Triethylene melamine has about the same action as does nitrogen mustard.[61] Colchicine has resulted in temporary improvement in some cases of mycosis fungoides.[68] (The author has under observation a man believed to have mycosis fungoides. He had relief of pruritus for four months from colchicine by mouth. It was then no longer effective.) ACTH and cortisone are temporarily helpful in some cases of lymphoblastoma. Some patients are made worse.[37, 55, 57]

DISCUSSION

It should be stressed that the clinical manifestations of these conditions, with the possible exception of mycosis fungoides, are not specific or diagnostic. The classification of a patient with lymphoblastoma, or even the diagnosis of lymphoblastoma, may be difficult and require prolonged observation.[9, 36, 46, 64] The difficulty is well illustrated by the case of a patient who was presented before a meeting of the Los Angeles Dermatological Society.[50]

The patient, a 9-year-old white boy, was first observed by the author March 17, 1951, because of a lesion "like a little boil" which had appeared on the left side of the nose two or three weeks previously. No pus was expressed from the lesion and no similar lesion had ever been present before. There was no history of injury preceding onset. Upon examination the lesion was observed to be a semi-hard erythematous nodule 1.2 cm. in diameter. The submaxillary nodes were not enlarged. The left anterior cervical lymph nodes were possibly enlarged, as was one in the left axilla. No abnormalities were noted upon examination of bone marrow. Results of examination of the blood and the urine were within normal limits. Only chronic inflammation was noted in microscopic examination of a left cervical lymph node that was removed April 2, 1951. The lesion was excised in toto April 12, 1951. A year later there was no evidence of recurrence.

It was generally agreed by those attending the presentation of the patient that the lesion was lymphoblastoma of some type. Winer[74] favored a diagnosis of reticulum cell sarcoma. Subsequently, the sections were studied by other dermatopathologists and pathologists, with the following diagnoses: Cutaneous lymphoblastoma, possible cutaneous reticuloendotheliosis, possible Letterer-Siwe disease, and probable cutaneous lymphoma.

Diagnosis and classification of these conditions are not helped by the fact that some patients show

characteristics of more than one of the lymphoblastomas. Keim[34] reported a case (Case 10) in which there was evidence of mycosis fungoides and lymphatic leukemia. In a case reported by Miller[45] (Case 4) the pathologic changes of Hodgkin's disease were noted in 14 lymph nodes, whereas in one node the structure was characteristic of small cell lymphosarcoma. Ginsburg[26, 27] found little biologic difference between lymphosarcoma and Hodgkin's disease, and pointed out that there is no clinical way of differentiating them. The histologic structure is not always diagnostic. Perhaps lymph node imprints[67] will help in differentiation. The observation that some lymphoblastomas apparently change from one type to another, and that transition forms exist between all the groups,[4, 23, 38, 39, 70, 73] does not help to clarify the situation.

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Film Library Has Record Year

RALPH P. CREER, secretary of the A.M.A. Committee on Medical Motion Pictures, reports that 2,088 medical motion pictures were lent to medical societies, medical schools, hospitals and other scientific groups during 1952. This represents an increase of 516 over 1951, and is the greatest number of films distributed since the A.M.A. motion picture library was started.

—A.M.A. *Secretary's Letter*

Regional Anesthesia for Office Procedures

CHARLES F. McCUSKEY, M.D., Los Angeles

• *Many minor surgical procedures can be performed successfully and safely with regional anesthesia in an office which is properly equipped.*

A careful history and physical examination should never be omitted. Oxygen with a breathing bag and mask and an ultra short-acting barbiturate for intravenous use should always be available and ready for immediate use.

When proper aseptic technique and the previously mentioned precautions are observed, the variety of diagnostic and therapeutic blocks and minor surgical procedures which can be performed in an office will depend upon the ability of the physician using them.

THE TYPE OF WORK that it may be necessary for a physician to do in his office depends to some extent upon the location and availability of hospital facilities, but the basic principles for the successful use of regional anesthesia must always be observed whether the procedure is done in the office or in a hospital.

Before any injection is started the patient should be quizzed to ascertain if he has any known idiosyncratic reaction to local anesthetic agents or other drugs.

Oxygen with a breathing bag and mask should always be available and ready for immediate use.

An ultra short-acting barbiturate for intravenous use should be readily accessible. Barbiturates do not prevent untoward reactions to local anesthetic agents but they will control the convulsions resulting from an overdose of procaine and thereby make it possible to keep the patient oxygenated until the toxic effects have worn off. Since the safer local anesthetic agents paralyze respiration well ahead of cardiac effects, if the patient is kept well oxygenated no permanent ill effects should result from an overdose or from accidental intravenous injection. In addition to the precautions already noted the following points should be adhered to:

1. Asepsis.

2. Aspiration must always be done before injection through a stationary needle.

3. Anesthetic solutions should be freshly prepared for each patient.

4. Epinephrine solution 1:1000 should be added to the anesthetic solution just before injection. Not more than six minims should be added to each 100 cc. of solution.

5. Epinephrine solution should not be used in patients with toxic thyroid disease or severe cardiac disease.

6. Adequate preliminary medication should be used to allay nervousness and fear. (If the patient is alone, this may be omitted in office procedures.)

7. Care should be exercised to avoid trauma to periosteum and bone.

8. Injections should be made extraneurally.

9. Operation should not be started until anesthesia is complete.

The local anesthetic agents commonly used are procaine, intracaine and metycaine. The toxicity of these agents is in the order named, procaine being the least toxic. It should also be remembered that 20 cc. of 2 per cent solution is more likely to produce an untoward reaction than 40 cc. of 1 per cent solution. The solution used, therefore, should be freshly prepared in each case and the strength of the solution used should not be greater than the size of the nerve to be blocked requires. For larger nerves, such as those in the brachial and sciatic plexuses, stronger solutions are necessary. Never should the strength of procaine or intracaine solutions exceed 2 per cent.

Untoward reactions are produced by the intravenous injection of the anesthetic agent or by the rapid absorption of the agent from a highly vascular area. The symptoms vary according to the dose. Following rapid absorption the pulse may increase or decrease; dyspnea, pallor, nausea with or without vomiting; cyanosis; cold, clammy skin and the usual manifestations of shock may follow. Following intravenous injection there may be a convulsion with temporary loss of consciousness, slowing of the pulse, and slowing or complete cessation of respiration. The treatment for either is: (1) 100 per cent oxygen, by artificial respiration if necessary; (2) intravenous administration of pentothal, nembutal or seconal; (3) stimulants, such as caffeine or cora-

Presented before the Section on Anesthesiology at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

mine; (4) intravenous infusion of plasma or dextrose or saline solutions.

The regional blocks most often called for in office procedures are those for minor procedures and diagnostic or therapeutic blocks. Before attempting any of these procedures it would be well to practice the proper method of raising a skin wheal which must precede insertion of a needle into the tissues. In raising a wheal anywhere on the surface of the skin, a minimum of pain will be produced if the bevel of the fine gauge wheal needle is held parallel to the surface of the skin and a slow extrusion of the solution is begun just before the needle makes contact with the skin. In other words, the needle is pushed through a drop of solution on the skin and the injection is continuous as the needle enters the skin. Pressure is maintained until a wheal of the desired size is obtained. A long 22 gauge needle is then attached to the syringe in order to produce a field block. It is pushed sharply through the wheal and then, as solution is slowly injected, is advanced beneath the surface of the skin. During this maneuver the bevel of the needle is kept downward. When the general area for a change in direction is reached, a wheal is made in the skin by injection from beneath into the lower layers of the skin. Done in this manner, the entire procedure is almost painless. Four such wheals with connecting lines of infiltration will block off the area to be operated upon.

For procedures about the face and scalp, either a field block or a nerve block may be used, the choice depending on the location of the area in which the operation is to be done. The infraorbital and mental nerves are the most accessible for blocking about the face and when they cover the entire field of operation it is a very easy procedure. The scalp receives its nerve supply from the trigeminal or the cervical plexus. From the supraorbital ridge to the vertex of the scalp, the nerves of supply originate from the trigeminal nerve and are distributed as the supratrochlear, supraorbital, zygomatico-temporal and superficial temporal nerves. In the back of the head, from the root of the neck to the vertex, the great auricular, lesser occipital, great occipital and least occipital nerves supply the scalp. All these nerves may be reached at some point on a band circling the scalp. Unless the location of each is known with great exactitude it is far better to place a band of interlocking deposits of solution along the "hat band" beneath the galea than to add to any preexisting trauma by searching for the elusive nerve trunks.

Stellate ganglion blocks, whether for therapeutic or diagnostic reasons are feasible from several approaches. The lateral approach probably entails less risk than some others. In the method used by the author the patient flexes his head slightly in order to loosen the muscles of the neck. The transverse process of the sixth cervical vertebra is then sought by palpation with the index finger. When it is located, the finger with pressure maintained is slid downward until the transverse process of the seventh cervical vertebra is felt. Pressure is then applied medially and posteriorly in order to displace the great vessels of the neck from the path of the injection. That done, the needle is advanced medially perpendicularly to the midline of the body and slightly posteriorly until contact is made with bone. Contact usually occurs after the needle has been advanced about one centimeter, since all of the softer, displaceable structures are retracted out of the path of the needle with the index finger of the other hand and held back until block is completed. It is important that the needle be kept well above the clavicle lest the dome of the lung be punctured.

Lumbar sympathetic blocks are best done with the patient in a prone position and with a small pillow under the hips. Wheals are raised 3 to 4 finger breadths from the midline directly opposite the upper edge of the vertebral spine. The 4-inch needle, detached from the syringe, is advanced perpendicularly until the point impinges on the transverse process. In passing it may be well to note that the lumbodorsal fascia is very tender and a few drops of solution may be required to render passage of the needle painless. To do this, a small amount of solution is deposited on the periosteum of the transverse process. The needle is then withdrawn until the point is just beneath the skin. It is then reintroduced with a cephalad inclination and passed over the upper edge of the transverse process. Given a medial inclination, the point of the needle will make contact with the side of the body of the vertebra. In this position the needle is advanced from 2 to 3 cm. past the point at which contact was made with the transverse process. With this technique injections are made of L2, L3 and L4 to block the vasomotor supply of the lower extremity.

Blocks of the upper extremities can be accomplished by brachial block or by median and ulnar blocks at the elbows, or each digit can be blocked individually. On the hand or fingers the initial wheal should be raised on the posterior surface and the needle advanced from there to the front. Care should be taken not to inject too much solution around the fingers. Pressure from too much solution, particularly when it contains epinephrine, can cause permanent damage such as would be produced by applying a tourniquet. Older people with preexisting vascular disease are particularly liable to difficulty of this kind.

With proper care and gentleness many things can

be done safely and comfortably in a physician's office. It should be emphasized, however, that all proper precautions should be observed to prevent untoward reactions and that preparations be made to treat them should they occur.

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Discussion by* FREDERICK LEIX, M.D., *Los Angeles

Especially to be stressed are the basic principles of regional anesthesia which have been outlined. One of these, skin preparation, has undergone revision in the past few years. It seems reasonable that the same treatment should be accorded the skin overlying the area to be anesthetized and operated on as that given the hands of the operating surgeon. A soapy vehicle containing hexachlorophene (G-11) serves well for this purpose. The lathering and rinsing with quantities of water should be pursued for several minutes. The type of antiseptic solution used to paint the skin thereafter should be aqueous rather than an alcoholic tincture, because alcohol destroys the effect of hexachlorophene.

In my own observation, few practitioners perform regional blocks of the sympathetic nervous system as office procedures. For those who do so there are two warnings I would sound. A possible candidate for lumbar sympathetic block is a patient with acute thrombophlebitis of the lower extremity. Frequently he is receiving dicoumarol as an anticoagulant agent. Deep bleeding may occur in such a patient as a result of needle trauma, even to the extent of massive retroperitoneal hematoma. A fairly common complication of stellate ganglion block is pneumothorax owing to puncture of the apex of the pleura and the cupola of the lung. The method described by Dr. McCuskey stresses high injection of the blocking agent as a safeguard.

There are two practical points which I should like to mention.

Finger lacerations are frequently treated in the office. An ideal anesthetic is achieved by blocking the digital nerve opposite the midpoint of the proximal phalanx with 1 per cent procaine solution. This anesthetic agent should never contain epinephrine, should be small in amount (1 to 2 cc. for each nerve), and should be used on one side only whenever possible. A bloodless field may be obtained by the use of a soft rubber urethral catheter applied as a tourniquet for a short time (5 to 10 minutes). It is possible to cause ischemic necrosis of the finger or toe if these precautions are not observed.

Small children are suitable candidates for the use of local anesthesia if they are well premedicated with phenobarbital to allay apprehension. The average child of two years may be given one grain of phenobarbital with safety. In case the lesion to be treated is a small lacerated wound, a cotton pledget soaked with 10 per cent benzocaine solution may be applied to the wound for five minutes. This will permit painless cleansing of the wound and surrounding skin prior to injection of procaine for anesthesia during suturing.

The Development of Specialization in Allergy

A Historical Review and a View Ahead

SAMUEL H. HURWITZ, M.D., San Francisco

•Clinical allergy as a special field of practice is a little more than twenty-five years old. The organized efforts of the two national societies for the study of allergy and the many county, state and regional groups of physicians interested in allergic diseases have served to bring all workers in the field together in pursuit of a common objective. However, the foundation stones for the specialty were laid by a number of astute clinical observers during the past hundred years.

This historical sketch aims to portray these men and their work, and points out how the introduction of the skin test as a diagnostic method has dominated the clinical approach to allergic diseases during the past half-century —and that the technique is gradually losing some of its significance. This changing emphasis from the older diagnostic procedures to other techniques is the result of the discovery of the new hormones, cortisone and corticotropin (ACTH). These hormones have presented another method of studying the mechanism of allergic phenomena in man. Finally, brief reference is made to the growing recognition of the significance of the psychosomatic factors in the management of the allergic patient and the influence of this and the other additions to knowledge on the training of the future generations of allergists.

On March 18, 1819, Dr. John Bostock,[4] an English physiologist and clinician, read a paper before the Royal Medical and Chirurgical Society of London on a Case of a Periodic Affection of the Eyes and Chest, in which he presented to the members the history and clinical symptoms of a seasonal affection which had troubled him since childhood.

In January 1922, the American Association for the Study of Allergy was organized in San Francisco, and held its first meeting in June 1923. In March of the same year, an organizational meeting was held in the City of New York, which resulted in the founding of the Society for the Study of Asthma and Allied Conditions. The amalgamation of these two organizations in 1943 to form the Academy of Allergy, and the founding during the previous year of the American College of Allergists, served to bring all workers in clinical allergy together in the pursuit of a common objective.

The hundred year period from the clinical observations of Bostock to the organized efforts of many students of allergy covers the major contributions which have led to the recognition of allergy as a clinical entity and the development of specialization in study and treatment of the condition.

It was Carlyle who expressed the view that history is the essence of innumerable biographies and that anyone who wishes to know the history of any period must know the men who made it what it was. Although medical history is concerned more with ideas than with biography, the acceptance or rejection of an idea, particularly prior to the development of the experimental method, was determined in no small measure by the personality and reputation of the physician who advanced the idea. The historical development of interest in allergy as a clinical subject bears out this point.

John Elliotson, a contemporary of Bostock, in a clinical lecture delivered at St. Thomas Hospital in London on March 31, 1831,[9] made the first definite suggestion that hay fever depends on the flower of grass and probably upon the pollen. When the lecture was delivered, Elliotson was at the height of his career as a teacher and consultant in London. His great industry, acknowledged abilities and prepossessing manners made it possible for him to forge ahead in London. A year after his graduation from Cambridge in 1821, he was elected physician to St. Thomas Hospital, where he became the most energetic teacher of the day. His lectures on hay fever are of particular interest to students of allergy because Elliotson was probably the first to mention the occurrence of dermatitis of the hands following the handling of the flower of grass. In referring to this observation, he wrote: "On handling the flower of grass, her hands always became instantly inflamed; therefore there is clearly in her skin a peculiar susceptibility of irritation from the flowers

Chairman's Address. Presented before the Section on Allergy at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

of grass." And he concluded, "I presume that the same morbid state exists in the mucous membranes."

This keen observer also anticipated by a quarter of a century Hyde Salter's classical description of hay fever and asthma following exposure to animal epidermal substances.[13] Of a woman patient sensitive to rabbits, Elliotson wrote that proximity to rabbits "produced a running at the nose and eyes and soreness of the upper lip; that if she went into a place where there were rabbits, it came on; and that if her husband came in after having shot a rabbit and threw it down near her, those effects were instantly produced."[9]

The great error of Elliotson's life was the espousal of mesmerism. In 1843, he published a pamphlet describing "Numerous Cases of Surgical Operations Without Pain in the Mesmeric State." His wards became filled with hysterical and excitable women, who were magnetized to sleep in order to try the effects of the new remedy, the fame of which had spread far and wide. Because of these activities, Elliotson fell into disfavor and with him the original and brilliant clinical observations on hay fever which he made. Knowledge of the underlying cause of hay fever was thereby retarded by almost fifty years.

In 1873, Charles Harrison Blackley of Manchester, England, published his "Experimental Researches on the Causes and Nature of Catarrhus Aestivus (Hay Fever)," and in 1880 appeared his more complete work entitled "Hay Fever, Its Causes, Treatment and Effective Prevention." Of his own case and the circumstances which led up to his ingenious experiments on hay fever, Blackley wrote: "I have, as I have previously said, suffered from hay fever for more than twenty-five years, but the exact time at which the disorder first commenced, I cannot now remember. The attacks lasted only a few days, and then declined rapidly; and they seemed then to me, to be in some way dependent upon the commencement of warm weather."[2]

The well-controlled experimental observations of Blackley and the accuracy of his deductions from them are indeed remarkable when viewed in the light of present knowledge of hay fever, particularly since his work was done before the controlled experiment had come into general use in medicine. Being subject to the disease, Blackley tested on himself the pollen of nearly one hundred different species of grasses and flowers, in the fresh as well as in the dried state and also, in some instances, in the form of alcoholic extracts. Five different ways of testing the pollen were tried: "(1) by applying it to the mucous membranes of the nares; (2) by inhaling it, and thus bringing it into contact with the mucous membranes of the larynx, trachea, and bronchial tubes; (3) by applying a decoction of the pollen to the conjunctiva; (4) by applying the fresh pollen to the tongue, lips, and fauces; (5) by inoculating the upper and lower limbs with the fresh moistened pollen."[2]

Thus it appears that Blackley anticipated by more than a quarter of a century the use of the diagnostic scratch and mucous membrane tests for pollen sensitivity. Of his use of the skin test, he wrote: "Whilst I was still suffering from my usual attack of hay fever, during the summer of 1865, as much pollen as could be obtained from two anthers of the Lolium italicum was applied to the center of the anterior surface of the forearm after the skin had been abraded, and to this the quantity of pollen named was applied after being placed on a piece of wet lint the size of the abrasion. This was covered with a piece of gutta percha, and the whole was held in position by a strip of adhesive plaster. The center of the other forearm was treated in exactly the same manner save and except that no pollen was applied to it. The scratching with the lancet raised a wheal such as is seen in urticaria or in the stinging with nettles. In a few minutes after the pollen had been applied the abraded spot began to itch intensely; the parts immediately around the abrasion began to swell, but this was apparently not due to any action on the cutis vera. In the above experiment the swelling seemed to be entirely due to effusion into the subcutaneous cellular tissues. The swelling attained its maximum in six hours, and then remained stationary for another eight hours. After this it gradually subsided, and in forty-eight hours, it had entirely disappeared. The arm to which no pollen had been applied did not exhibit any sign of swelling or irritation."[2]

Having established that seasonal hay fever is caused by pollen, Blackley undertook a series of experiments to find the quantity of pollen that may be floating in the atmosphere at low and high altitudes and the relationship between this quantity and the intensity of his own symptoms. After much experimenting on different methods, he decided on a procedure, of which he wrote: "Ultimately I was led to adopt a simple plan, which I afterwards found was recommended by Dr. Phoebus. This consists in the exposure of slips of glass to the open air for a given length of time, so as to allow any solid matter the air may contain to deposit upon the glass. Each slip of glass had a cell formed upon it with black varnish, so as to enclose a space one centimeter square. This square was coated with a thin layer of fluid prepared for this purpose. [In a footnote he stated that the fluid was made by mixing one part of water, two of proof spirit and one part of glycerine.] After being exposed for twenty-four hours, each slip was placed under the microscope,

and any deposit it contained was carefully examined, and the number of pollen grains counted."[2]

Other observations were made by attaching the glass slide to a kite which was flown at elevations of from 500 to 1500 feet. The pollenometric charts made by Blackley in 1866, 1867, and 1869 differ in no essentials from those of today.

In 1925, a half century later, Dr. William Scheppergrell of New Orleans, a pioneer student of hay fever, confirmed Blackley's observations on the pollen content of the upper strata by exposing pollen plates in an airplane at elevations over ten thousand feet[14]—studies which have led to the classical aerobiologic observations of O. C. Durham during recent years.

In his lifetime, Blackley, like many pioneers, was looked upon as somewhat of a faddist, and the fact that he practiced homeopathy caused his contemporaries to overlook his brilliant contribution to knowledge of the clinical aspects of allergy, thus again retarding development in this field by almost half a century.

The first noteworthy American contribution to knowledge of hay fever was made by Morrill Wyman of Cambridge, Massachusetts. With some members of his family he had been a lifelong sufferer of an autumnal form of the disease. In 1854, Wyman described the disease in his lectures at the Medical School of Harvard University, where he served for many years as Hersey Professor of the Theory and Practice of Medicine, a title which is still held by present incumbents of this position. In 1872, he published an exhaustive monograph on Autumnal Catarrh, the earliest contribution in the literature on ragweed hay fever. Of his experiment with ragweed, Wyman wrote: "Early in September 1870, I gathered in my grounds at Cambridge, Massachusetts, some Roman wormwood (Ambrosia artemisaefolia) in full flower, covered with pollen, taking the whole plant, stalks and roots. This was carried to the White Mountain Glen, about 1,200 feet above tide, where we remained till September 23 in the afternoon. The parcel containing it was then opened and freely sniffed by myself and son. We were both seized with sneezing and itching of the nose, eyes, and throat, with a limpid discharge. My nostrils were stuffed and my uvula swollen, without cough, but with the other symptoms of autumnal catarrh. These troubles continued through the night, and did not disappear till the afternoon following. Professor Jeffries Wyman (Professor of Anatomy in Harvard University), who was of the same party, but did not sniff the plant, had none of the symptoms just described."[18]

That pollen was an etiologic factor in hay fever, as was so ably established by the experiments of Blackley and Wyman, was by no means generally accepted in Europe and the United States, despite the established position of Wyman and the high regard in which he was held by his contemporaries. The reports by Blackley and Wyman were published at the beginning of the bacteriologic era—at a time when, owing to the influence of the researches of Pasteur and Koch, hay fever began to be considered an infectious disease. This theory found many adherents, although not one of Koch's postulates had been fulfilled in any of the experiments described.

In 1876, another monograph on hay fever appeared. It was written by Dr. George M. Beard, a well-known neurologist of New York City, a fellow of the New York Academy of Neurology and of the American Neurological Association. Beard, although well acquainted with the experimental researches of Blackley and Wyman, nevertheless concluded that the whole question of the origin and nature of hay fever was as yet an open one. He advanced the view that the disease, as well as the asthma which is frequently a complication, is essentially a neurosis—a concept of some importance in view of the stress placed in recent years on the psychogenic aspect of allergic manifestations. Owing to the great influence of Beard's writings on his contemporaries, more than a quarter of a century was to elapse before the significance of Blackley's and Wyman's work was fully appreciated.

VON PIRQUET AND THE FOLLOWING HALF-CENTURY

No historical review of the beginning of the present clinical concept of allergic manifestations would be complete without a brief sketch of the introduction of the skin test, which has so dominated the clinical approach to allergic diseases during the past fifty years. In 1906, Clemens von Pirquet, professor of pediatrics at the University of Vienna, who had acquired an international reputation for his classical work on serum disease, vaccination, and tuberculosis, suggested the term *allergy* for the changed reactivity of the organism following the repeated introduction of pathogenic substances. These studies led to the development of the tuberculin test, which was not only a new diagnostic method for the study of tuberculosis in childhood, but paved the way for the study of the pathogenesis of many other diseases and particularly those of allergic origin.

In 1909, the von Pirquet scarification technique was used by Henry Lee Smith[17] in the study of a patient sensitive to buckwheat, which he published under the title, "Buckwheat-Poisoning." When Smith reported this case at a meeting of the Johns Hopkins Medical Society, those who were students in the Johns Hopkins Medical School at the time (the author among them) little realized that this case

report opened a new approach to the diagnosis of allergic diseases. Smith not only obtained a positive reaction to a scratch test with buckwheat but produced a constitutional reaction in the patient. Dr. William S. Thayer, clinical professor of medicine at Johns Hopkins at that time, who suggested the test, Dr. Rufus Cole, later director of the Rockefeller Hospital, and Dr. Smith served as controls. In them the application of buckwheat to the scarified skin gave negative results.

The skin test as employed by Smith soon stimulated many other similar studies in this country. In fact, it may be claimed that the skin test as a diagnostic procedure in allergic diseases is essentially an American contribution.

In 1912, O. M. Schloss[15] used the cutaneous test in his studies on children sensitive to common foods, and a year later Clowes[6] obtained positive skin reactions in ragweed-sensitive patients. This pioneer work was followed by the studies of Goodale[10] who tested asthmatic persons who were sensitive not only to pollens but to horse dander; and Goodale's observations stimulated the extensive research on asthma carried out by I. Chandler Walker at the Peter Bent Brigham Hospital in Boston. Walker used the cutaneous test for the diagnostic recognition of a wide variety of allergic conditions owing to sensitivity to inhalants, foods and other allergens.

Other methods of testing for sensitivity soon were suggested. Smith had thought of testing buckwheat-sensitive patients by the conjunctival route, but at the suggestion of Thayer the cutaneous test was substituted. And Goodale was among the first to obtain mucous membrane reactions by the direct application of pollen extracts. Intracutaneous testing had been used by W. L. Moss at the Johns Hopkins Hospital preliminary to the administration of therapeutic sera, but the first employment of the intracutaneous technique in general allergic diagnosis is usually credited to Robert A. Cooke of New York City.

In 1911, Leonard Noon, working in Sir Almroth Wright's laboratory at St. Mary's Hospital in London, published a paper in the *Lancet* entitled, "Prophylactic Inoculation Against Hay Fever."[12] Noon, who died when but thirty-five years of age, had already achieved a reputation for his researches on tetanus toxin and antitoxin and other contributions to immunology, but it is for his pioneer work on the treatment of hay fever that he is best known to students of allergy. His name will continue in bright usage so long as the Noon unit for measuring pollen dosage remains the most practical method of measurement. It is of historical interest, however, that Karl Koessler, working at the Sprague Institute in Chicago, had anticipated Noon's work. In an article on "The Specific Treatment of Hay Fever (Pollen Disease)," published in Forchheimer's Therapeusis of Internal Diseases, Koessler wrote: "In May 1910, unaware of the work on this subject done in A. E. Wright's laboratory, I began active immunization against hay fever, and thus far I have treated forty-one patients by this method."[11]

Stimulated by the work of Noon which was later continued by John Freeman of London and Koessler in this country, numerous botanical surveys of hay fever producing plants were begun and extended to almost every region of the United States. Among the earliest of these surveys, and one of particular interest to students of allergy in California, was one carried out by Harvey M. Hall, formerly of the Department of Botany, University of California, and published in the Public Health Reports of the U. S. Public Health Service in 1922. This work was stimulated by the late Dr. Grant Selfridge, a San Francisco otolaryngologist, who in 1918 published one of the earliest reports in California on pollen desensitization.[16] Those who knew Dr. Selfridge, the author included, were impressed by his scientific curiosity, particularly since his training was essentially that of a surgical specialist.

With the publication of the successful therapeutic results in hay fever, the development of clinical applications of the accumulating lore of allergy proceeded at a great pace and many other allergic manifestations were treated by similar methods.

A. F. Coca and Robert A. Cooke contributed much to knowledge of the immune mechanism underlying allergic phenomena and the control of allergic disease by desensitization or, as they preferred to designate it, hyposensitization. Their studies, as well as those of W. W. Duke of Kansas City and Warren T. Vaughan of Richmond, Virginia, were among the first to stress the role played by other allergenic substances than pollen as a cause of allergic manifestations. Duke was the author of the first complete textbook on allergy published in this country.[8] The first edition appeared in 1925 under the title "Allergy, Asthma, Hay Fever, Urticaria and Allied Manifestations" and contained observations on the importance of foods, drugs and physical agents in allergic diseases. To Vaughan[19] clinical allergists are indebted for many pioneer investigations on other allergic conditions, such as migraine, and for his botanical classification of foods. His concept of allergic equilibrium has helped to explain some puzzling problems in the interpretation of symptoms of allergic origin.

Noon referred to his method of treatment as "prophylactic inoculation," and Koessler used the terms "specific treatment" and "active immunization." Despite the intensive research on the mech-

anism of desensitization during the past twenty-five years begun by Coca and Cooke, and since then extended by a host of workers, there is as yet no acceptable explanation of the mechanism underlying the allergic reaction and its control. The concept suggested by Dale and Laidlaw[7] in 1910, that histamine may be a participating factor in anaphylaxis, was soon used to explain the basis of allergic manifestations in man, a view which was greatly strengthened by the observations of Duke in 1923 and 1924 on urticaria caused by physical agents. The concept of allergy due to physical agents suggested by him has led to many investigations on the role of excessive histamine formation as a cause of allergic phenomena. These studies have stimulated investigations on many therapeutic methods designed to control excessive histamine release in the tissues of allergic persons. However, neither the use of histaminase, the anti-enzyme of histamine, nor desensitization against histamine has proved effective.

Whereas the introduction of the hormones, cortisone and corticotropin (ACTH) have dwarfed the many advances in the drug therapy of allergic diseases made during the past twenty-five years, no historical sketch would be complete without brief mention of other drugs which have been found helpful. Epinephrine, first isolated by Takamine in 1901 and later by Abel of Johns Hopkins, has been since its first use in asthma during the first decade of this century one of the most potent weapons. And when the experimental work of Chen[5] and co-workers in 1926 proved the value in bronchial asthma of the alkaloid, ephedrine, islated from a Chinese plant, Ma Huang, by Nagai in 1887, search was stimulated for other sympathomimetic drugs and the search has continued to the present day.

About a decade ago, another drug was added to the therapeutic armamentarium. Although Askanazy, as early as 1895, found that the salt of theophyllin produced beneficial effects in angina pectoris, its usefulness in combination with ethylene diamine, as aminophylline, was not fully recognized until the year 1940.

The addition of two other groups of useful drugs in the management of allergic patients is so recent as to require only brief mention. Their future place in the treatment of allergic diseases must await the verdict of more research and clinical experience. The antihistamine drugs have already proved to be valuable agents in the symptomatic management of patients with allergic disease, and the introduction of the antibiotics has served as a powerful weapon to combat the secondary infections of the respiratory tract that so frequently complicate allergic diseases.

No discovery, however, has so shaken the foundations of present-day ideas of the basic mechanism of allergic phenomena as the research, during the past several years, on the part played by the pituitary and adrenal glands in human hypersensitiveness. A historical sketch is not the place for a discussion of the role of the corticosteroids in the therapy of allergic diseases. Probably the greatest significance of the introduction of cortisone and corticotropin, remarkable as their effects may be on patients with allergic disease, lies not so much in the therapeutic results achieved as in the stimulus to basic research on the immunochemical mechanisms underlying allergic phenomena and on ways of modifying or perhaps of bringing about profound changes in the allergic constitution and in the adaptive processes so brilliantly postulated by Hans Selye.

There is another straw in the wind, which would appear to indicate that the care of allergic patients is fast passing beyond the period of technicology. The breeze seems to blow toward the ever-increasing appreciation by physicians of the vital part played by psychogenic factors in allergic diseases. Whereas the importance of these factors in such chronic ailments as hypertension, diabetes, peptic ulcer and many other chronic conditions has been fully recognized, little emphasis has been placed on the psychiatric management of patients with allergic disease. Many contributions to the literature on this phase of therapy have appeared in recent years, but considerable resistance has been shown by allergists to the adoption of psychotherapeutic methods. This aversion may owe (as Ross and Wilson pointed out in their chapter on Psychotherapy in Bronchial Asthma, published in Abramson's text on the Somatic and Psychiatric Treatment of Asthma[1]) to overemphasis placed by allergists on organic factors, and by psychiatrists on psychogenic factors. It is probable that either of these factors alone or both synergistically can act as trigger mechanisms in giving rise to allergic manifestations. In some instances symptoms may result from allergic causes alone, in others from psychogenic factors, but perhaps in the majority of instances both allergic and psychogenic mechanisms serve to bring about and prolong allergic manifestations. It would appear, therefore, that allergists of the future will need to devote more time to the psychotherapeutic as well as to organic factors in the management of patients.

Furthermore, the training of the next generation of physicians specializing in allergy will be influenced, in no small measure, by the ever-expanding horizons which in recent years have removed diagnosis and treatment of allergic disease from its limited technological confines. This trend will of necessity greatly influence not only the kind of undergraduate instruction in allergic diseases given in medical schools but also the planning of post-

graduate teaching. The postgraduate instructional courses on the many facets of allergy presented both by the American Academy of Allergy and the American College of Allergists fill a great need, and these organizations are to be commended for the vigor with which they have carried out this part of their program. However, there still remains some difference of opinion as to how instruction in allergy should be planned on the undergraduate level, and under whose aegis certification of the allergists should be placed.

It is common knowledge that undergraduate instruction in allergy in medical schools has lagged in spite of the fact that allergic phenomena constitute an essential part of the basic sciences of immunology, bacteriology, physiology, pathology, pharmacology, endocrinology and immunochemistry. Those who have had the responsibility of teaching medical students the fundamentals of allergy have been impressed with the immensity of the task of presenting adequately a subject which has such wide ramifications. It has become the conviction of many teachers of this subject that undergraduate instruction in allergy can be given most effectively as part of the teaching of the individual basic science. Thus a course in immunology or immunochemistry should include a discussion of sensitization in experimental animals and in man, and the fundamentals of the mechanism involved both in anaphylaxis as well as in human hypersensitiveness. The physiologist and pathologist could devote some time to the physiologic and pathologic aspects of the allergic reaction, and the pharmacologist to an evaluation of the common sympathomimetic drugs, antihistamines and the newer hormones, in the drug therapy of allergic diseases.

In brief, undergraduate teaching in allergy should be made a part of the instruction in the basic medical sciences. These basic facts and theories can then be coordinated with the common clinical manifestations of allergy such as hay fever, asthma, eczema, urticaria, gastrointestinal distress and allergic reaction to drugs. This integration of the subject can best be carried out in the out-patient allergy clinic by a member of the clinical staff adequately trained in this branch of internal medicine. Such a plan would lessen the trend, now so prevalent, toward overburdening the medical curriculum with instruction in the subspecialties. It must be admitted, however, that this is contrary to the program of the American Academy of Allergy and the American College of Allergists. Both of these organizations are on record as sponsoring undergraduate lecture courses in this specialty.

Closely related to the problem of undergraduate and graduate instruction in allergy is that of certification. In 1948, a joint committee representing the American Academy of Allergy and the American College of Allergists adopted a resolution[20] recommending the establishment of an independent or autonomous board. It is the conviction of many, however, that certification in allergy should continue to be the function of the boards of Internal Medicine and Pediatrics. The creation of an independent board might stunt rather than advance progress in knowledge of allergy, because it would deprive this subspecialty of the sustenance from the mother trunk upon which its growth must depend, and without which the dependent branches would undergo gradual atrophy.

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What is New in Adoption

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• Adoptions arranged through adoption agencies are on the increase because there are more agencies. The Citizens Committee on Adoption of Children in California has concluded that there are very few adoptable children under care in orphanages. Fifty per cent of physicians would prefer agency adoptions, but only 24 per cent actually refer both expectant mothers needing such services and couples wishing to adopt infants to such agencies. The program in this field of social welfare should be child-centered and physicians should seek and give cooperation to such agencies.

DURING the last three or four years there has been a significant increase in the number of adoption agencies in California and consequently in the amount of agency adoption services. In 1947-48 when the new law authorizing public as well as private adoption agencies went into effect, only one out of every seven children was being placed by the agencies. The remainder were placed through other sources. By last year almost one out of four children was placed through agencies—an indication that people will turn to agencies when services are available to them. At the end of January 1952, 1,100 children under care of adoption agencies in California were in their adoptive homes awaiting completion of adoption and another 1,300 children were being studied for placement. As the newer agencies get into full swing, greater coverage will be possible and the percentage of agency adoptions can be expected to continue to increase.

In many areas throughout California interested citizens and agencies are reexamining the present adoption program in order to provide better coverage and make changes to otherwise provide improvements in service. More than two years ago through the generosity of the Columbia Foundation and the Rosenberg Foundation a sum of money was assigned to activate and organize the Citizens' Committee on Adoption of Children in California.

This committee, a statewide organization, and county citizens' committees in 12 counties throughout California were organized to determine what the citizens of the state consider to be and will accept as a sound adoption program. In order to answer this question, the committees have considered all the facts, criticisms and differences of opinion relating to existing adoption practices and on this basis have come up with a summary and recommendations. This has already accomplished a great deal of good in clearing the atmosphere and in providing a better working relationship between the adoption agencies and the various professional groups interested in one way or another in adoption.

The agencies also are taking steps to examine their own programs to see what changes can be made. California adoption agencies meet periodically to exchange points of view, share experiences and plan programs designed to attain uniform standards and practices including more effective relationships with physicians and others in the community.

Following is a review of the current findings and criticisms of adoptions of children in California.

1. The supply does not meet the demand. For every baby available for adoption there are ten couples who want to adopt babies. A statement frequently made was that orphanages and foster boarding homes were filled with babies available for adoption. The Los Angeles County Committee conducted a study of all children under care in institutions and boarding homes, private and public. It was found that of 3,394 children under the age of 17 years whose cases were studied, only 105 were orphans in fact. Of 2,032 living in foster homes, 29 per cent were there because their homes were undesirable, 24 per cent because of the illness of a parent, and 16 per cent because the mother was employed.

Further analysis revealed 416 children who were rarely or never contacted by parents or relatives and who were therefore possibly adoptable, with difficulty. Two hundred twenty-nine of them were more than 12 years of age. Adoption agencies had started plans for adoption of 80 of the 187 who were less than 12 years old. It seems reasonable to conclude, therefore, that there is not, in fact, a number of adoptable children supposedly being "hidden out" in institutions.

2. Real progress is being made in attempts to determine why many adoptions are arranged individually and privately without the services of a licensed adoption agency. The most frequent criti-

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cisms are of "red tape," long delay and overemphasis on meticulous matching of child to foster parent. As to the "red tape," it must be said in defense of agencies that they are child-centered. They attempt to perform a service of high quality, and with limited funds they have difficulty in moving rapidly in home selection and placement. Placement methods have recently been changed to permit the earliest possible placing of numbers of infants within a few days to a few weeks after birth. A real effort is being made to place children with minor handicaps, physical or mental, and children handicapped by background. No longer does a social worker consider only a perfect child eligible for adoption.

3. Probably the greatest contribution to the field of adoption by the Citizens' Committee on Adoption has been a survey of the position of physicians in this social welfare problem. It has sometimes been said that social workers have not received or given cooperation in this phase of welfare planning. The evidence indicates that 50 per cent of physicians preferred agency adoptions, but only 24 per cent actually referred to social agencies both the expectant mothers with social problems and couples seeking to adopt children. Thus, three-fourths of the physicians interviewed arranged for placement of babies directly with foster parents in order to satisfy their own patients. In so doing they centered their consideration on the adoptive parents and not on the baby. Obviously there is urgent need for continued discussion in the hope that physicians and social workers may resolve their differences by close cooperation.

4. The problem of difficult-to-place children of minority groups, from the viewpoint of race and color, is being studied by the Los Angeles County Adoption Agency and considerable progress has been made in this direction. The Children's Home Society of California, the only statewide privately-supported adoption agency, is also working with this group.

5. Assistance to unmarried mothers is vitally needed. In the state of Washington the Children's Home Society has set up a program for the care of unwed mothers during pregnancy, delivery and the postpartum period. Physicians and hospitals are cooperating by rendering this service at a minimum fee. In California a similar project has just begun. This will be most important since, with this service available, there will be less reason for the so-called exchange arrangement in which care for the patient and the baby may be arranged by a third party.

6. Finances have limited the work of private as well as public agencies. The number of agencies charging fees has doubled in the last few years. One agency in California finances its program entirely through fees. The principle of a fee has even been written into law which established the county adoption agencies in California. It is expected that greater emphasis will be placed on this in the future. However, there are still differences of opinion as to whether or not the fee should be on a sliding scale based on ability to pay, or should be standard and sufficient to cover the cost of services rendered to the prospective adoptive parents, or sufficient to cover also part or all of the cost of service to the child and the natural parents.

It is recognized that the medical profession has a community responsibility as well as an ethical one. As such, it must take an active part, seek and give cooperation with the social welfare group. The adoption program must be child-centered, and the medical profession's aim should be to find the best homes for children—not just children for patients.

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Hyperinsulinism and Neuromuscular Disorders

A Consideration of the Association of Pancreatic Adenoma with Wasting States

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• *Five cases, one reported herein, have been described in which progressive generalized muscle wasting, muscle fasciculations, increased myotatic irritability, and progressive motor disability were in evidence. In all of these cases, a pancreatic islet cell adenoma was present. In four of them arrest of the symptoms of muscular disease followed upon surgical removal of the islet cell adenoma. In the other case the tumor was not observed until postmortem examination, and in that case there was also histologic evidence of widespread and severe degeneration in the anterior horn cells of the spinal cord.*

The observations give rise to conjecture upon the possibility that endocrine dysfunction plays a part in the genesis of progressive muscular atrophy.

NEUROMUSCULAR DISORDERS belong to a clinical group in which the cardinal signs and symptoms are related to progressive wasting of muscles. As a group these disorders are etiologically vague and pathogenically unspecific. The broader classification of neuromuscular disorders includes those states characterized by some demonstrable alterations in the anterior horn cells, or in their peripheral processes, or by some alteration in structure in the muscle fibers, per se. The term *progressive muscular atrophy*, or the alternative term *progressive spinal (or nuclear) muscular atrophy*, is usually employed to designate that group of muscle-wasting states in which the anterior horn cells are the site of demonstrable alteration (amyotrophy). Phenomena in progressive muscular atrophy, therefore, are progressive wasting of muscles, muscle fasciculations, increased myotatic irritability, electromyographic evidence of fibrillation, and progressive motor weakness, involving chiefly the truncal and appendicular muscles. In the most classical form of this disorder there is an inevitable progression of the wasting of muscles to the point of almost complete motor disability, bedridden status, and death.

Since knowledge of this disorder, as of other progressive muscle-wasting states, is meager, every clue as to etiologic or pathogenic factors seems worthy of pursuit. No clue has as yet been obtained, for example, as to the character of any toxic, viral, chemical, genetic, metabolic, or deficiency factors which presumably might be causative. Recently, however, implication that disordered endocrine function plays a part in progressive muscle-wasting states has become increasingly impressive as more and more clinical observations have been recorded.[2-6]

There seems sufficient reason, therefore, for reporting here in brief the study of a case of progressive muscle wasting in which an endocrine factor seemed responsible. In this case, there was a generalized progressive wasting of muscle, muscle fasciculations, electromyographic evidence of muscle fibrillation, increased myotatic irritability, and progressive motor disability. Perhaps of greater importance is that this presumably inexorable progression of muscle atrophy was arrested following the surgical removal of a tumor of the pancreas, histologically confirmed as an islet cell adenoma.

It is noteworthy that the literature contains reports of four other cases of this kind, all published since 1946. In three of the previously reported cases, removal of a pancreatic adenoma brought about an arrest of the signs relating to progressive wasting of muscles; and in the fourth case, in which a pancreatic adenoma was not discovered until autopsy, there was microscopic evidence of an associated severe and widespread degeneration of anterior horn cells of the spinal cord (amyotrophy).

REVIEW OF LITERATURE

Silfverskiold in 1946[5] reported upon two male patients, one 17 years and the other 34 years of age, who had progressive generalized wasting of muscles, muscle fasciculation, electromyographic evidence of fibrillation, increased myotatic irritability, and progressive motor disability associated with a pancreatic adenoma. In each case the progression of clinical signs was arrested by surgical removal of

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the tumor, histologically verified as an adenoma of islet cell type.

Lidz and co-workers[2] in 1949 reported the case of a 23-year-old man in whom there was arrest in the signs and symptoms of progressive wasting of muscles, muscle fasciculations, increased myotatic irritability, and progressive motor disability after a pancreatic islet cell tumor was removed. There were no sensory abnormalities noted and no other neurologic abnormalities.

Tom and Richardson[7] reported a case in which at postmortem examination of a 33-year-old woman who had had progressive generalized muscle wasting, not only was an islet cell tumor found, but severe and widespread degeneration of the ventral horn cells of the spinal cord (amyotrophy) was observed histologically.

REPORT OF A CASE

A 42-year-old white man, a salesman, was first observed in November 1950 with complaint of progressive weakness and wasting of the muscles and of a "quivering" feeling in the muscles for about one month. During the preceding week the patient had noted especial difficulties in climbing stairs, in walking more than a short distance, and in buttoning a shirt, holding a pencil, using a knife and fork. In the previous few days he had begun to note that his feet tended to "slap" after he had walked any considerable distance.

Upon inquiry the patient said that for the previous year he had been noting increasingly frequent transient attacks of weakness, associated with tremulousness, mild disorientation and confusion, "blurring" of vision and diplopia, most often on awakening in the morning or just before breakfast. He had found that taking food relieved the attacks, and eventually he drank sweetened orange juice for amelioration of symptoms. Several months after the onset of these attacks he consulted a physician who made a diagnosis of hyperinsulinism. At that time the content of sugar in the blood, repeatedly determined, was subnormal (30 to 40 mg. per 100 cc.). Pancreatic adenoma was suspected and laparotomy was carried out. The suspicion was not confirmed but approximately two-thirds of the tail of the pancreas was resected. The previously described attacks continued and within a month after the operation the patient noted the signs and symptoms of progressive muscle wasting.

At the time of examination by the author, approximately one month later, there was diffuse atrophy of moderate degree of the appendicular and truncal musculature, generalized muscle fasciculation, increased myotatic irritability, and electromyographic evidence of fibrillation. Walking was difficult and of the "steppage" type. Hand grasps were weak. Rising to a sitting or standing position was difficult. All deep reflexes were intact and hyperactive, except the Achilles reflexes, which were diminished. No sensory abnormalities were noted. All other neurologic findings were within normal limits.

During the next few weeks the patient continued to have transient early morning attacks associated with hypoglycemia (the sugar content of the blood was 6 mg., 15 mg. and 18 mg. per 100 cc. on three occasions). Muscle wasting and muscle fasciculation continued and motor disability increased. Surgical consultants concurred in a diagnosis of hyperinsulinism and recommended reexploration for possible pancreatic adenoma. In February 1951, four months after the onset of progressive muscle wasting, a pancreatic adenoma 2 cm. in diameter was removed from the inferoposterior surface of the head of the pancreas. It was histologically identified as an islet cell tumor.

No further early morning attacks or hypoglycemia were noted, and muscle wasting and motor disability were arrested. In the course of several weeks, with the aid of physiotherapy, the patient regained considerable motor capacity. Seven months after removal of the pancreatic adenoma, he was able to grasp a pencil, to write, to button his shirt, and to walk approximately a mile without tiring. He had regained nine pounds of weight lost during the illness.

DISCUSSION

The pathogenesis of the clinical entity commonly referred to as "progressive muscular atrophy" remains obscure. Duchenne (in 1848) and Aran (in 1850) expressed belief that the disease is of muscular (myogenic) rather than of neural (neurogenic) origin. In 1853 Cruveilhier reported that in such cases he had noted a slimness of the anterior roots of the spinal cord, and thus attention was focused on the possibility that progressive muscular atrophy was neurogenic; and in 1860 Luys reported that degenerative changes were observed in the anterior horns of the spinal cord at postmortem study of patients who had the disease. Within the next decade or two the work of Leyden (1876), Landouzy (1885), Dejerine (1885), and Erb (1891), served to bring about a clear distinction between progressive muscle atrophy related to anterior horn cell alteration (neurogenic) and muscle dystrophy associated with alterations in the muscle fibers, per se (myogenic). Since that time, although anterior horn cell alteration is a sine qua non of progressive muscular atrophy, the cause of anterior horn cell dissolution remains obscure.

It is well to consider, therefore, the possibility that disordered endocrine function plays a part in the disease clinically characterized by progressive wasting of muscles, muscle fasciculations, electromyographic evidence of fibrillation, increased myotatic irritability, and progressive motor disability.

The clinical features in all of the five cases reviewed herein were identical. In all of them the symptoms were associated with the existence of a pancreatic islet cell tumor.

The evidence is strong in support of a hypothesis that at least in some cases of progressive muscular atrophy, abnormality of endocrine function may bring about the sine qua non of this disorder—anterior horn cell dissolution (amyotrophy). And it gives rise to conjecture that either in the islet cells or in some remote endocrine gland (such as anterior pituitary, thyroid or adrenals) there might be sufficient hormonal imbalance or altered hormonal function to result ultimately in anterior horn cell dissolution. The well known hormonal antagonisms which exist between the islet cell hormone (or hormones) and those of the anterior pituitary (Houssay[1]) and

the physiologic interdependence of these and other hormone-producing organs needs no emphasis. As to the five cases reviewed here, there can be little doubt of endocrinic pathogenesis; in all of them the clinical features conformed to those of "progressive muscular atrophy" except that the progress was arrested by removal of an islet cell tumor. Further study is needed to determine to what extent pituitary-islet cell, pituitary-adrenal-gonadal, or pituitary-thyroidal, or other endocrine system dysfunctioning is of etiologic significance in progressive muscle-wasting states (amyotrophy).

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Stress Incontinence of Urine

A Consideration of Etiologic Factors in Women

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• *Urinary continence in women is intimately associated with adequate support of the neck of the bladder. Any increase in tone of the bladder musculature, or in the intravesicular pressure, substantially increases the liability to incontinence. Obstetrical trauma may cause stress incontinence by disrupting supports of the neck of the bladder and by stretching the fascial structures of the posterior portion of the neck of the bladder. Minor injury not grossly demonstrable may occur in this way and upset the very delicate balance existing between the forces of the detrusor muscle and the resistance of the urethrovesical junction.*

In correcting the defects associated with stress incontinence, there are specific indications for various methods—active exercise, plastic reconstruction of the bladder and urethral supports, and the various operations for suspension of the neck of the bladder.

URINARY STRESS INCONTINENCE, not an uncommon symptom, plays a large and distressing role in the daily lives of many women. Varying explanations for the lack of continence have been advanced, and there is no unanimity of opinion concerning the exact nature of the defect. A most disturbing aspect of the problem is that, in most series, measures taken to correct the condition are seldom successful in more than 85 per cent of patients. Any method of treatment which leaves one out of eight to nine patients no better off than before invites careful review and evaluation. A logical approach to consideration of the nature of stress incontinence should be to study normal continence and specific variations which accompany the defect.

NATURE OF THE DEFECT

Recent reports by Muellner,[20] Millin and Read,[17] and Marchetti[14] showed quite lucidly that normal urinary continence is associated with adequate support of the bladder neck. Using methods of fluoroscopic observation and cystography, they demonstrated that in continent primiparae the bladder has a smooth outline (Figure 1) with its base just above the symphysis pubis. When the subject is standing, coughing or straining the bladder maintains its regular outline across the base, while when the subject is voiding the bladder base descends in such a way that the region of the sphincter becomes the most dependent portion of the bladder and assumes a pointed appearance (Figure 2). It is felt that this descent of the internal sphincter opens it slightly and allows urine to enter the proximal urethra, which is followed by a reflex contraction of the detrusor muscle, and micturition ensues. When voiding is voluntarily stopped, the neck of the bladder is pulled up to a level slightly higher than it was originally, the bladder base becomes horizontal, the detrusor relaxes, and the urinary stream is shut off. The same situation holds with continent multiparae, except that the bladder base sinks to a lower level on standing or straining. However, the regular horizontal outline of the bladder base is maintained until voiding is initiated.

Anatomically it is quite apparent that the region of the neck of the bladder (or internal urethral orifice) is closely supported by the pubocervical portion of the endopelvic fascia, by the pubococcygeus portion of the levator ani, and to a degree by the deep transverse perinei muscles.[5] From the position of these muscles it is seen that one of their functions would be to raise and to allow lowering of the bladder neck, thus playing an active role in the voluntary initiation and cessation of voiding. They would also resist any sharp increase in intra-abdominal or intravesical pressure and thereby protect the internal urethral sphincter against stress.

It has been observed cystographically that when a women with urinary incontinence reclines, the bladder base assumes a normal position and outline (Figure 3). When she assumes the erect position, even without straining, the bladder base takes on a pointed shape and the region of the internal sphincter becomes the most dependent part of the bladder (Figure 4). It then takes only a minimal descent to open the internal sphincter and allow urine to escape.

The factor of pressure relationships within the

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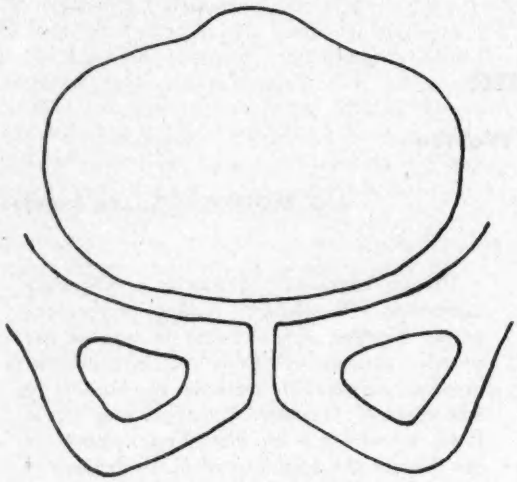

Figure 1.—Diagram of bladder in nullipara. Note good support and even outline of the bladder base. (After Muellner.)

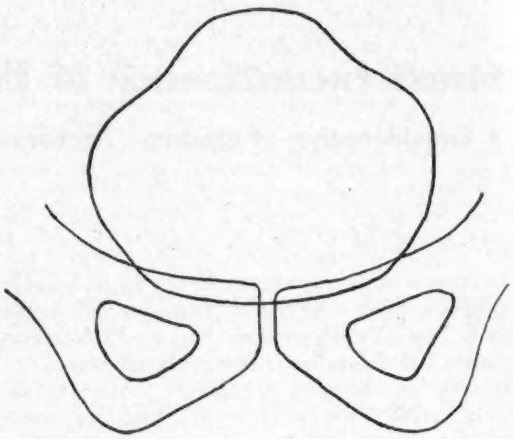

Figure 3.—Incontinent multipara reclining. Note regular outline of the bladder base. (After Muellner.)

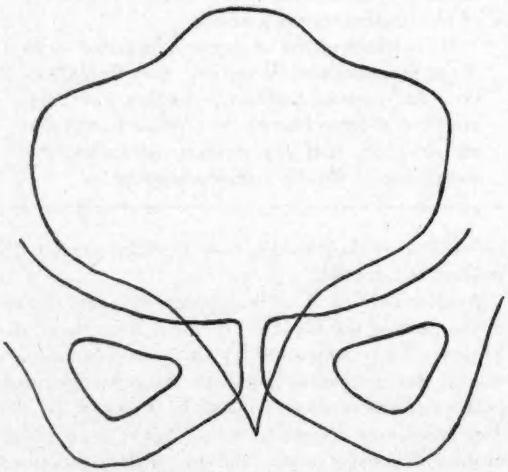

Figure 2.—Nullipara. Note appearance of the bladder base and descent of the internal urethral orifice with voluntary attempt to void. (After Muellner.)

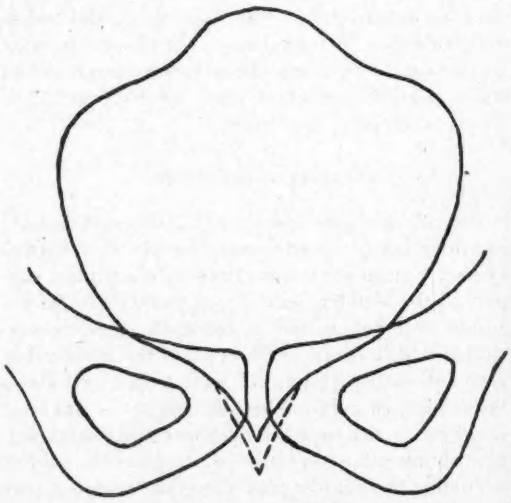

Figure 4.—Incontinent multipara standing. Note the pointing of bladder base and descent of the internal urethral orifice. The dotted line indicates the minimal additional descent necessary to initiate voiding. (After Muellner.)

bladder is also important in considering continence. With the bladder at rest, the intravesical pressure is usually measured between 0 and 15 centimeters of water.[6] During the course of active micturition, the pressure rapidly builds up to 50 to 100 centimeters of water until the detrusor muscle relaxes. It is logical that with the vesical neck or internal sphincter so constructed that it must counteract only a very low pressure when the detrusor muscle is relaxed, any increase in the resting tone of the bladder musculature would substantially increase the chances of incontinence. That this is true is shown by the increased incidence of incontinence usually found in association with such conditions as descensus uteri, pregnancy in the third trimester, and large pelvic tumors—conditions that are also known to be associated with increased intravesical pressure.[2, 7] In this regard, reflex irritability of the detrusor from ureteral stricture, or from trigonitis, might easily overcome the resistance of the sphincter.[7, 22] Consequently, in investigation to trace etiologic delineations of stress incontinence, cystometric studies to estimate the balance of the detrusor against the internal sphincter are considered useful.

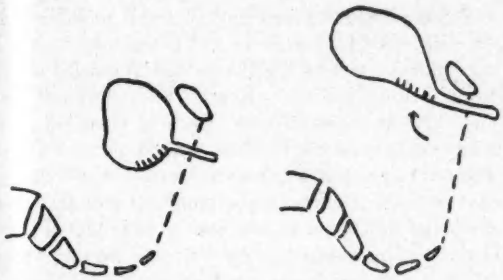

Figure 5.—*Left*, position and relationship of urethra and urinary bladder in pregnancy before engagement of the fetal head. *Right*, note compression of the urethra against the symphysis, and rotation of the base of the bladder into line with the urethra, both occurring with deep engagement of the head in labor.

ETIOLOGIC FACTORS

The nature of the trauma usually responsible for stress incontinence has been the subject of considerable debate. The condition is generally ascribed to trauma of the urethral sphincter resulting from childbirth. Some investigators attribute it to adhesions around the urethra which distort the sphincter and impair its function,[11] while others ascribe it to obstetrical injury of the supports of the bladder. Still another view is one of serious doubt as to whether obstetrical trauma is responsible.[19]

In an effort to evaluate this problem of obstetrical trauma in the causation of incontinence Malpas, Jeffcoate, and Lister[13] carefully studied the bladder and urethra by cystographic methods in various stages of labor. They demonstrated quite well that the neck of the bladder did not rise during labor but instead came to occupy a position closer to the symphysis. Also they found that the urethra did not elongate during labor but appeared to do so because the bladder base rotated (with the internal urethral orifice remaining stationary as a pivot) in such a way that the urethra and bladder base came to lie in the same straight line, making it difficult to indicate where bladder ended and urethra began (Figure 5). They were unable to show any relationship between the formation of a lower uterine segment and the position of the bladder as a whole; throughout labor the position of the urethrovesical junction and the bladder base remained unchanged. From this they would surmise that an extreme rotation of the bladder base from its normal horizontal position to the vertical position stretches the fascial investments of the bladder base and, more specifically, those of the posterior aspect of the bladder neck. After delivery, they postulated, the fascia does not recover its strength and the normal perpendicular relationship of the urethra to the bladder is lost. They expressed belief that minor injury, not grossly demonstrable, may occur in this way and upset the very delicate balance existing between the force of the detrusor muscle and the resistance at the urethrovesical junction.

Kennedy[11] attached considerable importance to the internal sphincter and stated that its action is augmented in large part by the voluntary action of the anterior portions of the levator muscles. He felt that, for the sphincter to function properly, it must be circular in configuration and free of adhesions. The trauma of childbirth, he believed, often injures the neck of the bladder, therewith producing adhesions about the sphincter and disrupting its fibers. Other investigators tend to doubt the existence of a sphincter as such, believing as do Denny-Brown and Robertson[6] that the internal sphincter is an integral part of the detrusor muscle. These investigators demonstrated that no relaxation of the sphincter may occur without a corresponding contraction of the detrusor muscle.

Muellner[19] was impressed by the fact that when signs of relaxation are discernible, there is no correlation between the extent of relaxation and the degree of incontinence. He was impressed, too, by the observation that pronounced stress incontinence occasionally occurs even though the pelvic supports are intact, and conversely by the maintenance of satisfactory continence in the presence of obviously poor bladder and urethral supports. Also, Jones and Kegel found that they could not predict which patient had stress incontinence and which did not when the patient was examined with the bladder empty. In the obstetrical and gynecological clinic of the University of Southern California School of Medicine, stress incontinence was present in only 41 per cent of patients with obstetrical sequelae severe enough to necessitate reconstructive operation. In a series of patients (with cystocele, uterine prolapse, etc.) Muellner was unable to establish more than vague correlation between the degree of stress incontinence and the parity of the patient or the degree of difficulty she experienced in labor. He was impressed by the inconstant time relationship between the trauma and the ensuing symptoms and he suggested that incontinence might be owing to acquired irritability of the detrusor muscle that permitted minor stimuli (as in coughing or sneezing) to produce contraction of the vesical musculature with a corresponding relaxation of the internal sphincter and, therewith, urinary leakage.

Kegel[10] stated that exertional incontinence resulting from obstetric trauma is the result of two factors: the actual laceration and separation of muscle fibers and fascia, and the separation of motor endplates from the muscles of pelvic support. He held that the latter results in a relaxation and attenuation

of supports over a period of time, which may result in stress incontinence. Kegel cited the ability of muscle to become innervated, even in adult life, and expressed the belief that active exercise of these muscles increases the muscle tone and hastens the process of reinnervation.

METHODS OF CORRECTION

Many varieties of procedures have been used to correct stress incontinence, including such methods as suburethral injection of paraffin, injection of sclerosing solution, massage and electrical stimulation, torsion of the urethra, and advancement of the urethra.[25] As would be expected from the foregoing discussion, the best results followed procedures aimed at supporting the neck of the bladder. Kelly's method of plicating the urethra and the neck of the bladder with fine silk usually is successful in 70 to 80 per cent of patients.[9, 3] As the procedure is extended to reconstruct more carefully all of the pelvic supports the results become better.[11, 4] However, it is the usual experience that as the length of the follow-up increases, so does the failure rate. In an effort to attain successful continence in patients not aided by the conventional reconstruction of pelvic supports, operations have been devised to support the region of the bladder neck in other ways. The Martius bulbocavernosus interposition operation utilized tissue from the labium majus, interposing it between the bladder and urethra above, and the vagina below. The Goebel-Stoekel-Frankenheim principle of supporting the urethra with the anterior rectus fascia has been modified by Miller,[16] Aldridge,[1] and Studdiford.[25] Millin[18] has so modified this procedure as to accomplish it entirely from above. Shaw[24] utilized fascial strips from the thigh, attaching them to the urethra and bladder neck, bringing them out through holes drilled in the pubic bone, and suturing them to the tendinous tissue of the adductor longus muscles. The operation of urethrocystopexy (suspending the urethra and region of the bladder neck to the symphysis pubis) has been advocated in this country by Marshall, Marchetti, and Krautz,[15] and in Europe by Perrin,[21] and Labry and Charvet.[12] It is not to be doubted that these more extensive and extravaginal procedures do lead to the cure of many patients left incontinent after a complete vaginal procedure. However, with few exceptions,[24] they too sometimes fail. In many cases the eventual failure is attributed to a mistaken diagnosis of the cause of incontinence, or to the technical inability of carrying the procedure to its correct conclusion. The point is clear, however, that none of these procedures has a place in the therapy of urge incontinence, passive incontinence of urinary fistulae, or urethral incontinence of neurogenic origin.

The nonoperative treatment of stress incontinence was suggested by Davies in 1938[4] and advocated as a therapeutic test by Rashbaum and Mandelbaum in 1949.[22] However, it was Kegel[10] who effectively applied and demonstrated the practical value of muscular restoration by exercise with a perineometer. The necessity of sustaining the treatment (in 40 per cent of cases it takes longer than two months) and the belief that good results will be maintained only if the patient continues the exercises, would seem minor drawbacks in view of the report of success in 93 per cent of patients.[8] Jones and Kegel expressed the opinion that operation for relief of stress incontinence should not be done except in the case of prolapse outside the introitus. Their report of cure in 82 per cent of patients who previously had been subjected to reconstructive vaginal operation seems especially significant.

DISCUSSION

A review such as this, citing as it does the reasons for the diversity of opinion concerning the cause and mechanism of stress incontinence, at the same time indicates important factors to be considered before corrective therapy is begun:

1. There is a delicate balance between the intravesical pressure established by the detrusor muscle and the resistance that the urethrovesical junction is able to provide. In fact, it is felt that the internal urethral sphincter is an anatomical and functional part of the detrusor mechanism and that they each are incapable of independent action.

2. In the great majority of cases of stress incontinence it can be demonstrated that the region of the urethrovesical junction occupies the bottom of a funnel-shaped area of relaxation of the base of the bladder when the patient is standing or straining, or that there is a significant increase in detrusor tone.

3. Injury to the tissue in the region of the neck of the bladder, without gross abruption of the bladder supports, may be sufficient to cause exertional incontinence.

4. This defect may become apparent for the first time as the patient reaches, or is past, the climacteric period, and it would seem that atrophic changes in the pelvic supports, in addition to previous obstetric trauma, are important factors.

5. Other defects in urinary continence, attributable to inflammation (trigonitis), tumors, or nerve lesions (multiple sclerosis, spina bifida, tabes dorsalis), usually will not respond to therapeutic methods aimed toward supporting the neck of the bladder.

In light of these factors, if there be any doubt as to the nature and cause of urinary incontinence in a patient, cystoscopic and cystometric studies should

precede any attempt at correction. The choice of therapy then would naturally be consistent with the attending physician's experience and preference. In general, it would seem logical to employ the following principles in selecting therapeutic procedures:

1. If stress incontinence is unassociated with a disturbing cystocele, rectocele, or uterine prolapse, the methods of active exercise of the muscles concerned with pelvic support, as outlined by Davies and by Kegel, are indicated.

2. If a disturbing relaxation of the bladder, rectum, or uterus accompanies stress incontinence, a thorough support of the bladder neck should be incorporated into the vaginal plastic procedure designed to improve the relaxation.

3. If exercises and a thorough vaginal procedure do not relieve true stress incontinence not of neurogenic origin, one of the bladder neck suspension operations (fascial sling or urethrocystopexy) is in order.

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Bronchogenic Carcinoma in San Diego County

Relation of Mortality Rates to Findings in Mass Chest X-Ray Survey

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• *Nearly half the population of San Diego County was examined by chest x-ray for bronchogenic carcinoma. The disease was correctly diagnosed in 20 persons, of whom 17 died. Twenty-four others in whom it was not detected in survey films died of the disease in the following two years.*

Of the 20 cases found, 16 were in men, all more than 54 years of age, and of the 24 who died after "negative" classification, 20 were men over 40.

The death rate for men over 40 years of age from bronchogenic carcinoma is about one in a thousand. Because of the frequently rapid progress of the disease after onset, and the poor prognosis after the appearance of symptoms, x-ray examination every six months for men over 40 should be considered.

IN FEBRUARY 1950 a metropolitan mass chest x-ray survey was completed in San Diego County. Of a population of 556,800, almost half—245,061*—were examined by 70 mm. films and records were tabulated on 239,609. This presentation is an analysis of findings from that survey as related to the mortality from bronchogenic carcinoma in 1950 and 1951.

INCREASING INCIDENCE

The earliest figures available from San Diego city and county on the incidence of bronchogenic carcinoma are for 1920. In that year, two of 117 deaths from cancer were attributed to bronchogenic carcinoma, a proportion of 1.7 per cent and an incidence in the general population of 2.6 per hundred thousand. In 1930, the proportion was 3.5 per cent and the incidence 5.3 per hundred thousand; in 1950, 9 per cent and 11.1 per hundred thousand. This increasing incidence parallels the experience in the United States as a whole where, in 1948, the death rate from bronchogenic carcinoma per hundred thousand of population was 11.3, whereas in 1920 the rate was 1.1.

Part of the above computations was based on the 1950 mortality tables for San Diego County, in which 4,630 deaths were recorded.† Of these deaths, 683 were attributed to cancer, 62 to bronchogenic carcinoma. More important, in males of 45 years and older, there were 52 deaths from bronchogenic carcinoma, an incidence of 77.6 per hundred thousand among the 67,200 in that age group. In 1950, 52 males and 10 females died of bronchogenic carcinoma; in 1951, 58 males and 7 females (Table 1). In the two years combined, the median age group was 60 to 64 for males and 65 to 69 for females. It is notable that 86.6 per cent of the persons who died of bronchogenic carcinoma were males.

CASES FOUND IN SURVEY

Bronchogenic carcinoma was correctly diagnosed through the 70 mm. survey films in 20 persons; 24 others whose films had been classified as "negative" died in 1950 and 1951 of bronchogenic carcinoma. This total of 44 cases is an incidence of 18.1 per hundred thousand persons processed in the survey. Of the 20 cases found through the survey, final diagnoses were obtained from the patients' physicians by the survey's follow-up system in only 12. In the other eight cases the diagnosis did not accord with that made on the basis of the survey film or the patients were lost to follow-up until the death certificates carried the presumably correct diagnosis of bronchogenic carcinoma. The proportion of cases found in the survey, then, was 8.3 per 100,000 participants, close to the 7.8 per 100,000 for a similar survey of 536,072 persons in Boston.[4]

More important than the general case-finding average is the fact that 16 of the 20 cases found were in men, who were all over 54 years of age (Table 2). As the number of men more than 54 years old screened in the survey was 20,837, the proportion in this group was 76.92 per 100,000.

*This was 81.7 per cent of the 300,000 estimated eligible for x-ray survey in the San Diego metropolitan area.

†Includes deaths of San Diego County residents who died elsewhere in California.

From the Tuberculosis Control Service, San Diego Department of Public Health.

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TABLE 1.—*Bronchogenic Carcinoma as a Cause of Death by Sex and Age Groups, San Diego County (Including City).

Age	Combined 1950 and 1951 Male	Female
25 to 29	1	....
30 to 34	....	....
35 to 39	1	1
40 to 44	2	1
45 to 49	9	....
50 to 54	8	1
55 to 59	23	4
60 to 64	22	1
65 to 69	18	5
70 to 74	14	....
75 to 79	8	3
80 to 84	1	1
85 to 89	3	....
Total	110	17
Total, per year	127	

*Includes San Diego County residents who died elsewhere in California.

TABLE 2.—*Mass Survey Participants with a Final Diagnosis of Bronchogenic Carcinoma, by Age and Sex.

Age	70 mm. Positive Male	Female	70 mm. Negative Male	Female
25 to 29	....	....	....	....
30 to 34	....	....	....	....
35 to 39	....	....	....	....
40 to 44	....	....	1	....
45 to 49	....	1	2	1
50 to 54	....	1	2	....
55 to 59	4	....	7	1
60 to 64	4	....	4	1
65 to 69	4	2	1	....
70 to 74	3	....	1	....
75 to 79	1	....	1	1
80 to 84	....	....	1	....
85 to 89	....	....	....	....
Total	16	4	20	4
Total, male and female	20		24	

CASES MISSED

Against this high proportion is the fact that 20 cases in males over the age of 40 were missed in the survey from a total of 47,296 screened in those ages, a proportion of 42.55 per 100,000; and in the age group 55-59 years, seven men who were classified as "negative" in the survey died within two years of bronchogenic carcinoma—a rate of 114.7 per 100,000 in the 6,124 men in that age group who were screened.

OUTCOME OF CASES

As Table 3 indicates, the time from detection of the neoplasm in the 70 mm. film to death averaged nine months—six months for males and thirteen months for females. Five patients were asymptomatic at the time of the 70 mm. film; three of them (see 1706598, 1652005 and 1200997 in Table 3) underwent pneumonectomy at one, two and three months, respectively, after the discovery of the lesion by means of the film, and all three had metastases at the time of operation.

Death certificates are on record for 17 of the 20 persons in whom bronchogenic carcinoma was diagnosed on the basis of survey films. One (1107324) was alive and well in April 1952; on the same date another (1104068) was reported working, though very dyspneic. The third survivor (0111841), who had an adenocarcinoma proven by bronchogenic biopsy, refused operation and was lost to follow-up.

Of the 12 bronchogenic carcinomas pathologically classified, eight were squamous cell carcinomas and four were adenocarcinomas. In the other eight cases the type of lesion was not specified.

The author reviewed the 70 mm. films of the 24 persons who were classified "negative" in the survey but died of bronchogenic carcinoma. The impressions gained were that at least six of these persons should have been recalled for 14x17-inch films; that four of the six could have been classified as neoplasm suspects on the basis of the 70 mm. films alone (0512543, 0718217, 1910692, and 1005573 on Table 4), and that two of the four (0718217 and 1005573) may have been asymptomatic at the time the films were made. The detrimental effect of a "false negative" report, perhaps dissuading a person who otherwise might seek further investigation of symptoms, cannot be overlooked.

DISCUSSION

For the persons found "negative" in the survey who later died of bronchogenic carcinoma, the average time from the screening to date of death was 16 months. Most important, the average time before appearance of symptoms was only nine months. As only 10 per cent of patients with bronchogenic carcinoma can be cured after appearance of symptoms,[1, 2] a repetition of the 70 mm. filming a year later, while it might well have indicated the disease, would have been of little practical value.

The conclusion, therefore, is that six months would be a safer interval for roentgen screening of males over 40. Moreover, the author agrees with Pendergrass[3] that for adequate survey examination, films must be made on inspiration, on expiration and laterally, and must be of optimum quality—quality being more important than size.

A program entailing surveys every six months should not receive lip service unless there is a possibility of putting it into effect. Although the proportion of failures here recorded may justifiably lead to question as to whether mass attempts at detection of neoplasm by photofluorography should be abandoned, it is possible to contemplate a program whereby the necessary studies would be made on every man over the age of 40. In San Diego, where the tuberculosis case-finding operation of the Department of Public Health taxes the staff and budget

TABLE 3.—*Mass Survey 70 mm. Positive Bronchogenic Carcinoma Cases.*

Survey No.	Sex	Age	Survey 70 mm. Date	Symptoms at Survey Date	Operation: Date	Operation: Type	Death Date	Duration per Death Certificate	Interval from 70 mm. Film to Death: Months	Interval from 70 mm. Film to Death: Days†	Type of Bronchogenic Carcinoma	Autopsy
1201254	M	67	12-18-49	1 mo.	----	Died before operation	2- 2-50	Not stated	1	25	Epidermoid. Died of coronary thrombosis.	------
1706598	M	56	12-28-29	No	1-28-50	Pneumonectomy*	1-31-50	Approx. 2 mo.	1	3	Adeno.	Yes
1652005	M	64	1-21-50	No	5- 3-50	Pneumonectomy*	5- 3-50	Not stated	3	12	Not specified	Yes
1408857	M	64	1-11-50	6 mo.	No	No	9-21-50	Not stated	8	10	Not specified	No
1909993	M	68	1-19-50	9 yr. (cough)	No	No	10-25-50	6 mo.	9	6	Epidermoid	No
0308032	M	60	12-30-49	1 yr.	6-21-50	Thoracotomy*	8-19-50	12 mo.	7	19	Epidermoid	Yes
0111841	M	58	12-29-49	2 mo.	No	Refused	Moved out of area 1951 to L.A. County	------	----	----	Adeno. (Bronchoscopic biopsy.)	------
1104068	M	67	12-15-49	Beginning	3- 9-50	Pneumonectomy	Alive 4-23-52	------	----	----	Epidermoid	------
0300344	M	72	12- 6-49	6 mo.	2-27-50	Pneumonectomy	11- 2-50	Not stated	10	26	Epidermoid. Died of myocardial failure.	No
1107324	M	59	1- 5-50	14 yr. (asthma)	5-23-50	Pneumonectomy	Alive and well 4-24-52	------	----	----	Epidermoid	------
1200997	F	48	12- 7-49	No	2-27-50	Pneumonectomy*	7-30-50	7 mo.	7	23	Epidermoid	No
0502103	F	54	12- 9-49	No	11-21-51	Biopsy cervical lymph nodes	12-26-52	24 mo.	26	17	Not specified	No
1707713	F	67	1- 6-50	No	No	None	10-29-50	Not stated	9	25	Adeno. (Ref. 1)	Yes
0211541	F	69	1-21-50	Beginning	5- 1-50	Thoracotomy*	9- 9-50	9 mo.	7	19	Adeno. (Ref. 2)	No
0201901	M	77	12- 8-49	Beginning	No	None	10- 7-50	Not stated	10	----	Not specified. (Ref. 3)	No
1803081	M	65	12- 8-49	3 weeks	No	None	9-13-50	10 mo.	9	5	Not specified. (Ref. 4)	No
1307043	M	73	1-12-50	1 yr.	No	None	9-13-51	1 yr.	1	20	Not specified. (Ref. 5)	Yes
0908069	M	64	1- 9-50	2 yr.	9-48	Pneumonectomy	8-30-50	2 yr.	7	21	Not specified. (Ref. 6)	No
0400097	M	70	12- 6-49	Not stated	No	None	2- 5-50	1 yr.	2	----	Epidermoid. (Ref. 7)	Yes
0500772	M	56	12- 7-49	Yes	No	None	1-11-50	9 mo.	1	8	Not specified. (Ref. 8)	No

* Metastases at time of operation.

†Average time interval: 70 mm. film to death, 9 months; average, males, 6 months; average, females, 13 months.

1. Closed in survey follow-up as SN 360-1236 (healed primary tuberculosis) on diagnosis by private M.D., April 12, 1950, after survey review board had made diagnosis of "rule out neoplasm of lung" on January 31, 1950.

2. Closed in survey follow-up as SN 361-190 (chronic pneumonitis) on diagnosis by private M.D., April 7, 1950. Survey diagnosis on February 9, 1950 was (1) questionable pneumonitis, (2) questionable tuberculosis, moderately advanced, (3) elevation of diaphragm.

3. Closed in survey follow-up as SN 360-8XX (neoplasm of lung, type undetermined) June 7, 1950. Surgery had been advised against by private M.D. because of age. Was a neo suspect prior to survey.

4. Closed in survey follow-up as SN 360-1232 (pulmonary tuberculosis, moderately advanced) on diagnosis by private M.D.

5. Closed in survey follow-up June 8, 1950 as SN 370-1X0 (pleurisy) by county hospital diagnosis.

6. Diagnosed in September 1948 when he had pneumonectomy for bronchogenic carcinoma. Detected in survey with contralateral lung neo.

7. Entered Navy hospital before receiving report on 70 mm. findings.

8. Diagnosed originally in July 1949, 6 months before survey by 70 mm. film by San Diego Department of Public Health. Detected by survey prior to treatment.

TABLE 4.—*Persons with "Negative" Diagnoses on 70 mm. Films in the San Diego Mass X-Ray Survey Who Died of Bronchogenic Carcinoma in 1950 and 1951.*

Survey No.	Sex	Age	Duration per Death Certificate	70 mm. Date	Death Date	Interval from 70 mm. Film to Death: Months	Interval from 70 mm. Film to Death: Days	Interval from 70 mm. Film to Onset of Symptoms: Months	Interval from 70 mm. Film to Onset of Symptoms: Days	Autopsy
0512543*	M	64	3 months	1- 3-50	3-27-50	2	24	0	6 (minus)	Yes
0507513	M	64	Not stated	12-17-49	9- 8-50	9	1	Unknown		Yes
1303446†	M	59	3 months	12-17-49	10-17-50	10		7		No
1701293	M	53	Not stated	12- 6-49	11- 8-50	11	2	Unknown		Yes
0718217*	F	45	3 months	1-16-50	9-12-50	7	26	4	4	Yes
0171650	M	64	2 months	1-30-50	6-10-50	4	10	1	20	No
1909172	M	45	Not stated	1-14-50	9-22-51	20	8	Unknown		Yes
1910692*	M	80	Not stated	1-21-50	11-12-51	21	20	Unknown		Yes
0408954	M	59	6 months	12-21-49	7-28-51	19	7	12	23	No
1208141	M	66	3 months	1-20-50	5- 1-51	15	11	11	19	No
1005573*	M	56	18 months	12-16-49	8- 8-51	19	22	0	8	Yes
0310853	M	63	4 months	1-20-50	6-11-51	16	21	11	9	No
1409819	M	77	Not stated	1-18-50	8-31-51	19	13	Unknown		Yes
0508931	M	55	6 months	12-20-49	9-15-51	21	5	14	25	No
1908781†	M	42	24 months	1-11-50	9- 4-51	19	23	4	7	No
0705971‡	M	55	1 month	12-13-49	6- 9-51	17	26	15	4	No
0605985‡	M	55	7 months	12-16-49	3-21-51	15	5	7	25	Yes
1411112	M	71	12 months	1-30-50	9-25-51	19	25	6	5	No
0112773	M	53	3 months	1- 5-50	8- 8-51	19	3	15	27	No
1808645	M	47	4 months	1- 3-50	11-30-51	22	27	17	3	No
1507682	M	57	6 months	1-13-50	4- 6-51	14	23	7	7	Yes
1306905	F	79	Not stated	1-11-50	10-11-50	21		Unknown		No
0808989	F	56	6 months	1-24-50	3- 2-51	13	8	6	22	Yes
0503843	F	61	Not stated	12-10-50	4-12-51	16	2	Unknown		Yes
			7 months average			16 months average		9 months average		

* Grossly positive on review. †14 x 17 indicated. ‡Previously clipped and not available for review.

to screen 70,000 70 mm. films and patients annually, it would be necessary, for ideal neoplasm case-finding, to make three films on each of 67,200 males twice yearly, or a total of 403,200 additional films. This, naturally, would be a staggering burden and out of balance with more important health department activities. Yet it is difficult to expect that many of the 67,200 men would spend $30 a year for prophylactic screening by private physicians.

These statistics should stimulate thoracic surgeons, who have the greatest knowledge and experience in the diagnosis, treatment and prognosis of bronchogenic carcinoma, seriously to consider devoting the amount of time they now spend on such activities instead to the study of this disease. With such talent thus employed, it would seem safe to predict that soon there would be better diagnostic tools than x-ray films of the chest and better therapeutic weapons than pneumonectomy. The fact that man has two lungs may have retarded, temporarily, such a search.

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NOTE: Mimeographed copies of more extensive tabulated data used in the preparation of this presentation may be obtained from the author.

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CASE REPORTS

- **Prolapse of Gastric Mucosa Through the Pylorus**
- **Traumatic Rupture and Avulsion of the Diaphragm**
- **Cytomegalic Inclusion Disease in an Adult**

Prolapse of Gastric Mucosa Through the Pylorus

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PROLAPSE OF REDUNDANT gastric mucosa is a definite clinical entity which has come to be better recognized during the past decade. Radiologists are calling attention to it with increasing frequency.

According to Rees,[7] Von Schmieden reported the first case in 1911. Melamed and Hiller[6] reviewed the literature in 1943 and found only 19 cases reported. Since then the condition has been noted in a fairly large number of cases. It has been reported observed in as many as 7.7 per cent of patients examined roentgenographically by gastrointestinal series, although that figure is considered rather high. Scott[9] stated that it was noted in 1.04 per cent of roentgen examinations of naval personnel with gastric complaints. The large discrepancy between those two reports of incidence can probably be attributed to the fact that the average age of naval personnel is relatively low, whereas the condition seems to occur more frequently in the fourth and fifth decades of life.

Following is a report upon a patient with pronounced prolapse of gastric mucosa who was recently observed by the authors.

REPORT OF A CASE

A 41-year-old white woman had had "indigestion" for five years with such symptoms as bloating, sour stomach, cramps, diarrhea and occasional bloody stools which the patient attributed to hemorrhoids. In September 1950 she began having substernal pain every day. It started after breakfast and lasted all day. Upon roentgen examination of the gastrointestinal tract a duodenal ulcer was noted. Dietary measures resulted in great improvement. X-ray examination was carried out again in January 1951 and although there was no evidence of ulcer there was a polypoid mass in the lower portion of the stomach and pylorus. Exploratory operation was advised.

The patient then came under the authors' observation, and on January 25, 1951, another x-ray examination was carried out.

The roentgenologist reported: Fluoroscopically, moderate hypertrophy of the mucosa of the cardiac section was noted. There was increase in peristalsis in the distal portion of the stomach. The rugal markings extending through the pyloric canal were greatly accentuated. A space-consuming lesion within the outline of the duodenum had the appearance of a polypoid or pedunculated lesion within, but not obstructing, the cap (Figure 1). At six hours the stomach was empty.

Figure 1.—Film of barium-filled duodenum with appearance of a space-consuming lesion.

The blood count was normal.

Laparotomy was done February 11, 1951. There was a soft, spongy diffuse mass in the prepyloric region. It was easily pushed through the pylorus into the duodenum and it seemed to fill the entire prepyloric area. There was no evidence of ulceration. The mass was thought to be prolapsed gastric mucosa, and owing to the extensiveness of the lesion partial gastrectomy with retrocolic anastomosis was performed.

The pathologist reported a moderate degree of low grade hypertrophic gastritis with prolapse of the mucosa through the pyloric sphincter.

The patient recovered and had no recurrence of symptoms.

ETIOLOGY

Several theories have been advanced as to the cause of mucosal prolapse. Eliason[2] believed that a low grade inflammatory process in the lower third of the stomach causes hypertrophy of the mucosa, which in some cases leads to prolapse. This view was corroborated by Haworth and

Rawls[4] in a discussion of prepyloric gastritis, a process limited to the gastric mucosa, which becomes thickened and may prolapse through the pylorus. However, they considered the gastritis as a psychosomatic disorder in which the parasympathetic system is subjected to excessive stimulation of central nervous system origin.

Rees[7] attributed the condition to a resistant narrowing of the pylorus, causing hyperperistalsis which loosens the attachment of mucous membrane. Recently, Melamed and Melamed[5] reported four cases in which prolapse of the mucosa existed simultaneously with congestive heart failure. In two of the cases the diagnosis was made radiologically and confirmed at autopsy; in the other two the diagnosis was also made by roentgen study, but with the clearing of the congestive failure, the prolapse also disappeared so far as could be determined by fluoroscopic and roentgen film examination. The Melameds concluded that edema of the mucosa from congestive heart failure may cause redundancy and prolapse.

Scott[9] advanced the theory that prolapse results from excessive and abnormal mobility of the prepyloric mucosa on the muscularis, and that the activating factor is hyperperistalsis owing to neurogenic or chemical stimuli.

Symptoms

Prolapse of gastric mucosa into the duodenum causes symptoms of so wide a variety that diagnosis on the basis of clinical observation is extremely difficult. In general, symptoms are not severe, unless there are complications, but they are of sufficient intensity to cause patients to seek medical advice. Cramping pain and intermittent epigastric distress are the most common symptoms. The distress may be in the form of fullness, bloating, belching and heartburn. Pain is fairly constant. It is centered in the epigastrium, but may radiate to the costal margins or to the back, or as in the case herein reported, it may be substernal. Nausea alone or with vomitus is next in frequency. Hematemesis and melena may be present if there is ulceration. Gastric acidity is not distinctive. Anorexia, anemia and loss of weight may be prominent symptoms. In short, the symptoms are such as those that are also associated with many other gastric or duodenal disorders, and frequently with gallbladder or liver disease.

Pathology

Usually there is redundancy of mucosa of the lower end of the stomach with hypertrophied rugae. The mobility of the mucosa on the muscularis is greater than normal. The pyloric muscle becomes hypertrophied. There may be evidence of local gastritis. In the case reported herein, the mucosal folds were very redundant and could easily be pushed through the pylorus for a distance of 5 cm.

Diagnosis

As the clinical symptoms are not distinctive, diagnosis is made by radiological studies. To be noted roentgenographically is a filling defect of the duodenum observed as a lobulated mushroom-shaped area of translucence with a central thin streak of barium, and intact mucosa. The translucent area may vary in size, shape and appearance during a single examination. There is no evidence of irritation of the duodenal bulb, but gastric peristalsis is hyperactive in most cases. Mucosal prolapsus is most difficult to differentiate from prolapsed pedunculated gastric tumors and polyps.

Complications

Complications sometimes occur:

(a) Ulceration, which apparently occurs quite frequently and may cause the prolapsus to be overlooked.

(b) Hemorrhage, which may follow minor erosions of the mucosa or actual ulceration.

(c) Gastric retention, because of variable pyloric obstruction.

(d) Malignant changes occurred in at least one case.[8]

Treatment

In mild cases conservative therapy is indicated. This consists of a regimen similar to that prescribed for patients with ulcer, including the use of mild antispasmodics and an antacid preparation if needed. In most cases the condition will be well controlled by this treatment.

In severe or progressive cases, operation must be considered. Many different surgical procedures have been advocated, but not enough data have been obtained as yet to permit adequate evaluation of the relative advantages of each.

Some of the operations employed are partial gastrectomy, pyloroplasty, gastrojejunostomy, simple excision of redundant mucosa with or without pyloroplasty, and anchorage of the mucosa to the muscularis. The authors believe partial gastrectomy and excision of the redundant mucosa with pyloroplasty are the better procedures.

Indications for operation are: (1) Persistent pain after a long period of adequate medical treatment; (2) hemorrhage; (3) obstruction of the pylorus.

SUMMARY

Prolapse of gastric mucosa through the pylorus is a definite clinical entity with symptoms very similar to those of peptic ulcer or allied gastric disease. The clinical manifestations may suggest the diagnosis, but the principal diagnostic aid is roentgen study.

In most cases conservative treatment gives satisfactory results. Operation is indicated only when symptoms do not clear up with medical management, or when a large tumor-like defect is observed in roentgen study, or when complications such as hemorrhage or obstruction occur.

A case in which operation was done is reported herein.

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Traumatic Rupture and Avulsion of The Diaphragm

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RUPTURE of the diaphragm, or severance of the fibers of this muscle, should be distinguished from avulsion or separation of the diaphragm from the wall of the chest. Although both conditions are traumatic in origin and may have similar consequences, the symptoms, signs and laboratory findings being indistinguishable, the procedures necessary for repair are usually dissimilar.

Diaphragmatic rupture or avulsion has been attributed to a multitude of causes, varying from sneezing to an airplane power dive. Traffic accidents are prominent as a cause of this unusual injury; others are crushes, stabs, blows, bullets, a spicule of fractured rib, coughing, and straining during bowel movement. It is conceivable that congenital weakness may sometimes be a predisposing factor.

ANATOMY

The diaphragmatic muscle converges from its circumference to the large central tendon, into which it is inserted. The posterior origin is strong and tendinous, being part of the anterior longitudinal ligament of the vertebral column, and is not so likely to be avulsed or split. On each side the diaphragm originates from the lumbocostal arches or arcuate ligaments, and further laterally from the inner surfaces of the lower six ribs and costal cartilages. The anterior origin is from the posterior xiphoid region.

The important structures which pass through or behind the diaphragm are well protected by tendon or by decussating muscle bundles and are usually not injured with the diaphragm. These are the inferior vena cava, esophagus, aorta, thoracic duct, sympathetic and splanchnic nerves, and azygos and hemiazygos veins.

DIAGNOSIS

Although symptoms vary somewhat, the classic signs of shock are usually present in varying degree, the patient lying with knees drawn up against the chest, faint and perspiring or even in collapse. There may be pain in the upper part of the abdomen and left shoulder. The severity of trauma and of concurrent injuries will, of course, affect these symptoms.

Physical signs, in addition to those of shock, are the result of diminished vital capacity, mediastinal shift and thoracic displacement of the abdominal viscera. Such signs may not appear for hours when the viscera are slowly displaced into the thorax. Diminished chest excursion, dyspnea and cyanosis are present to greater or less degree, as are rapid thready pulse, diminution or absence of breath sounds on the left (sometimes with splashing or tinkling noises), and resonance on percussion changing to dullness as fluid accumulates. If the stomach is in the chest and is dilated, tympany is usually notable, sometimes as high as the third rib. Typically, the left upper abdomen and the lower chest are tender, the abdomen rigid. Often the abdomen is scaphoid, becoming progressively more so as abdominal viscera are displaced into the chest. The cardiac apex is usually displaced to the right.

Laboratory findings are of no aid in specific diagnosis, except for the following observations which may be made roentgenographically.[1]

1. Presence of abdominal viscus, such as stomach or colon, in the chest. (Often a gas bubble above a fluid level may be visualized in the organ.)

2. Invisibility of the injured dome, owing to concentric contraction on either side of the tear. Blood clots might also conceal the dome.

3. Intermittent twitching (seen by fluoroscope).

4. Pneumoperitoneum occuring when the abdomen is not openly wounded.

5. Pneumothorax occurring when the thorax is not wounded, but a hollow viscus is penetrated.

6. Absence of accumulated blood from the thorax in the presence of extensive injury of the lung with atelectasis and pneumothorax (evidence that blood has escaped through an opening in the diaphragm).

It is important to remember that roentgenographically visible changes may not appear for some hours after injury and that x-ray films and fluoroscopy should be repeated if indicated by physical signs and if the patient's condition is not definitely improving.

TREATMENT

Surgical operation is, of course, necessary for treatment of diaphragmatic injury, and as in any severe injury the patient must be brought to the best possible condition before operation. Sufficient time may be taken to overcome the initial shock, to administer fluids, blood, sedatives and antibiotics, and to empty the stomach by nasal catheter. The latter procedure is very important, for in many cases the stomach becomes enormously distended with fluid while angulation of the gastroesophageal junction prevents vomiting.

The authors prefer the transthoracic or the combined abdominal and thoracic approaches for operation, as with the abdominal approach it is difficult to replace the abdominal contents and hold them in place while the diaphragm is being repaired, especially since it is necessary to work high up under the costal arch.

If the transthoracic approach is used, anesthesia is administered intratracheally. Then the patient is turned to the unaffected side (almost always the right), and the eighth interspace is incised or the ninth rib resected.

The spleen is usually found in the thorax. It should be carefully inspected for tears in the capsule or at the hilus, and splenectomy, if indicated, should be performed at this stage. The other abdominal viscera should be inspected, repaired if injured, and replaced into the abdomen. Intra-abdominal pressure may make the replacement and retention of these organs very difficult. In one case, the authors brought the intestines out onto the anterior abdominal wall through a rectus incision to permit repair of the diaphragm.

It is usually advisable to crush the phrenic nerve. The pulmonary ligament is usually found to be torn, but need not be repaired.

Repair of laceration of the diaphragm often is very simple, interrupted non-absorbable sutures of cotton or silk being inserted in two rows. The use of fascia lata, or heavy sutures, does not appear to be necessary. It is conceivable that if there is loss of substance of the diaphragm, replacement by tantalum gauze might be required.

Replacement of avulsed diaphragm is somewhat more difficult. Tension must be applied to hold the diaphragm against the thorax until it can be made fast. Simple sutures cannot be relied on to hold, and there are no strong fascial structures to which the diaphragm can be attached. The authors found that mattress sutures looped through the

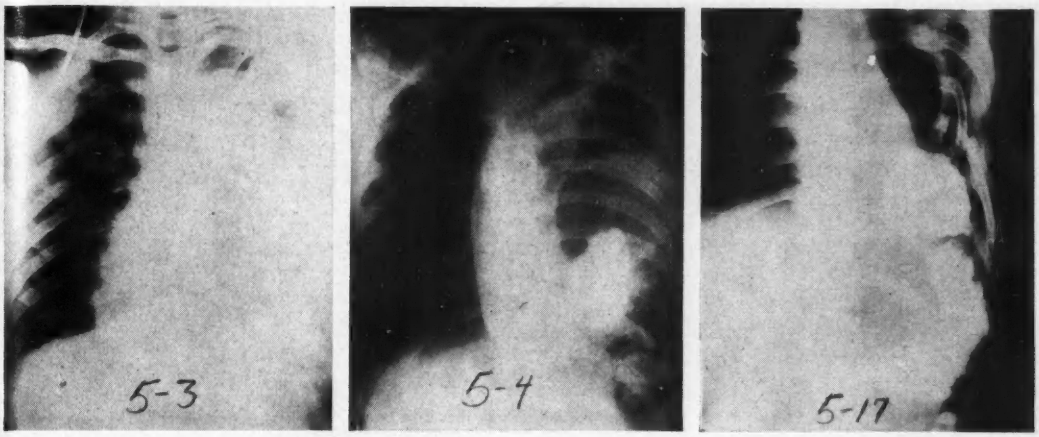

Figure 1 (Case 1).—*Left,* Taken two days after entry (preoperative), showing almost complete opacity of the left side of the chest with some mediastinal shift to the right. *Center,* preoperative film after barium ingestion, showing esophageal dilation, intrathoracic stomach. *Right,* twelve days after thoracotomy, showing a fistula from the splenic flexure to the left side of the chest.

whole thickness of the chest wall except the skin and subcutaneous tissues, some sutures being passed around a rib, would effectively hold the diaphragm abutted tightly against the wall. No. 2 catgut was used for these sutures, and all sutures were placed before any were tied.

For repair of the esophageal hiatus, one or two sutures should catch the external coat of the esophagus or stomach.

Chest closure is done in layers and a drainage tube is brought out through a stab wound to an underwater seal.

Postoperatively, oxygen should be given while dyspnea is present. Nasal suction is applied, and fluid and electrolytes must be replaced. Blood, proteins, vitamins and antibiotics are administered as indicated. Deep breathing, removal of bronchial secretions and early ambulation are important.

CASE REPORTS

Case 1: A 24-year-old man was admitted to the Monterey County Hospital shortly after an automobile accident, with multiple lacerations and abrasions, open fractures of both tibiae, and fracture of the left mandible. He was rational and in only moderate shock. The pulse was regular, the rate 130. Systolic blood pressure was 110 mm. of mercury, diastolic 80 mm. No injury of the chest was noted. With the patient under general anesthesia, the fractured tibiae were debrided, the fractures reduced and casts applied.

On the following day the patient was dyspneic and cyanotic, with temperature 102° F., pulse rate 152, respirations 24 per minute, and systolic blood pressure 76 mm. of mercury, diastolic 58. He was nauseated and vomited several times.

The left side of the chest was dull to percussion and breath sounds were absent, but the apex was hyperresonant, and high-pitched tinkling sounds could be heard. The heart was so displaced to the right that the maximum impulse was at the right sternal border. The left side of the chest was completely opaque in an x-ray film, which showed mediastinal shift to the right (Figure 1). No fluid was withdrawn on thoracentesis.

Dyspnea, vomiting and fever continued through the third day in hospital, and tympany and hyperresonance were noted over the upper half of the left side of the chest. On the following day, despite several transfusions of whole blood, the patient's condition became steadily worse. On x-ray examination, in addition to the mediastinal shift, the diaphragm appeared to be elevated to the level of the second interspace. After a small barium meal, the stomach was seen to be in the thorax, the esophagus dilated because of the pronounced angulation of the gastroesophageal junction. The roentgenologist believed that the diaphragm was elevated above the stomach (Figure 1).

Because of the gastroesophageal obstruction, dyspnea, cyanosis and pronounced mediastinal shift, it was decided to explore the thorax. The preoperative pulse rate was 140, the blood pressure 120 mm. of mercury systolic and 60 mm. diastolic. An anesthetic agent was administered intratracheally. An incision was made through the full length of the seventh interspace (the eighth rib was resected later in the operation).

The diaphragm was found to be avulsed from the left anterior and lateral walls of the chest, the tear extending from the paravertebral gutter almost to the sternum. The stomach, the transverse colon, the spleen and several loops of small intestine were crowding into the thoracic cavity. The stomach was greatly distended and congested; it appeared to be obstructed, and the venous circulation was impaired. There were two small lacerations of the spleen. The pulmonary ligament was torn. Two thousand five hundred cc. of serous fluid was aspirated from the chest cavity (this yielded no growth on culture). The spleen was removed and the phrenic nerve was crushed. The stomach, the small intestine and the colon were replaced into the abdominal cavity, where they were held in place with some difficulty.

The principal injury was true avulsion of the diaphragm from the wall of the chest. The difficulty in repair was that the endothoracic fascia was not firm enough to hold sutures from the edge of the diaphragm. Therefore, continuous sutures of No. 2 catgut were looped through the entire thickness of the chest wall except the skin and were reinforced by interrupted mattress sutures of the same material passed through the seventh interspace.

It appeared that the chest was thus completely closed from the abdominal cavity. It was later learned that a knuckle of colon at the splenic flexure either was caught in the suture or subsequently slipped up through a small rent, and became strangulated. The latter seems more likely, as great care was used in keeping the abdominal contents

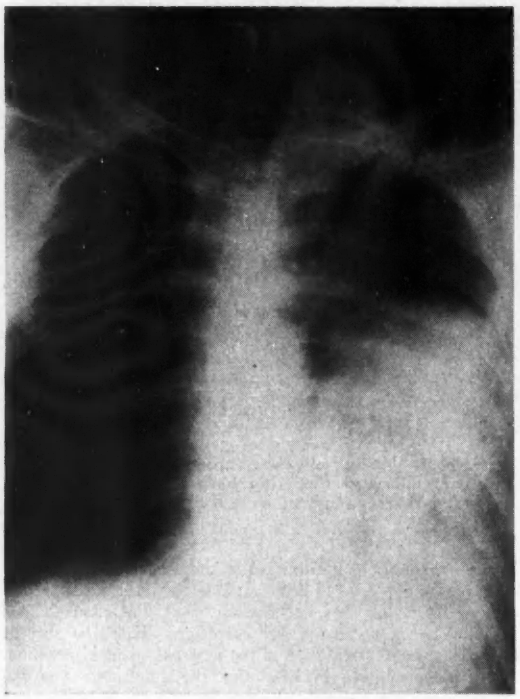

Figure 2 (Case 2).—Opacity of the lower two-thirds of the left side of the chest.

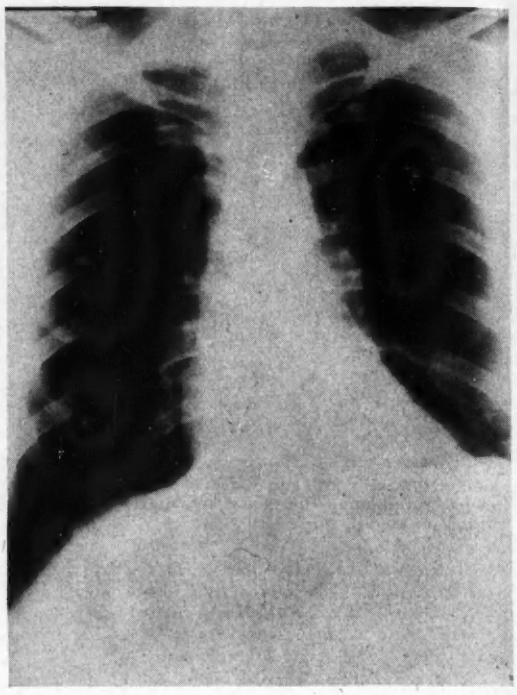

Figure 3 (Case 2).—Film taken six months after operation, showing a normal chest.

from the suture line. It is entirely possible that a small part of the edge of the diaphragm either was not sutured securely or subsequently pulled away from the wall of the chest.

A routine chest closure was done and a water-sealed drainage tube was placed through a stab wound in the posterior axillary line. Wangensteen gastric suction was instituted.

The patient's condition improved during the next few days; dyspnea and cyanosis diminished considerably. The intrathoracic tube drained poorly and was removed after 48 hours.

Dyspnea returned by the fifth postoperative day; it was relieved by removal of 2,300 cc. of turbid fluid from the left side of the chest. On the following day, x-ray examination showed that the mediastinum had shifted well back toward the left and the lung was expanded 60 per cent.

On the seventh postoperative day, 300 cc. of air and 150 cc. of fluid, fecal in appearance, were aspirated from the left side of the chest. On the ninth postoperative day, 6 cm. of the left sixth rib was resected. There was gas and about 500 cc. of fluid, apparently fecal, in the chest. A gray fibrin-covered empyematous cavity was opened and packed. Two days later the gastric suction tube was removed and the patient tolerated liquids taken by mouth.

Twelve days after the first operation a barium enema was given and on x-ray examination a fistula between the splenic flexure and the left chest was observed (Figure 1). On the same day transverse colostomy was done to divert the entire fecal stream.

Although the patient's condition gradually improved, fever continued as high as 100° F. Twenty-four days after the first operation, a left oblique subcostal incision was made. The colon was found adhering to the left posterior diaphragm, with a small opening extending through the diaphragm. The splenic flexure was resected and end-to-end anastomosis was done.

Six days later a large area of pneumothorax developed on the left side and the patient became very ill for a few days. A week later a small abscess in the subcostal incision was opened. Improvement was gradual while the chest wound continued to drain a small amount of seropurulent fluid.

Convalescence was slow. The fractures healed satisfactorily. The artificial anus was closed when the patient had been in hospital about three months. The chest and abdominal wounds healed, the latter very slowly. The patient was discharged six and a half months after admission.

Case 2: A 38-year-old man, thrown clear from an overturning automobile, fell prone on the highway. On admission to the Monterey County Hospital, he was unconscious and cyanotic. Respirations were rapid and grunting. The pulse was good, the rate 112. The systolic blood pressure was 128 mm. of mercury, the diastolic 84. In addition to multiple minor abrasions and contusions, there was a laceration of the right brow. The left pupil was dilated and reacted sluggishly to light.

The trachea was shifted to the right. The upper two-thirds of the left thorax was dull to percussion, the lower third flat, particularly on the lateral aspect. No breath sounds were heard on the left side. Peristalsis was occasionally heard laterally, just above the flattened area. There was an abrasion over the left costal margin. No abnormalities were noted in the right lung, and the abdomen was flat and soft.

On x-ray examination the left anterior diaphragm was seen to be elevated to the level of the second rib, the cardiac shadow displaced to the right. There was a large air bubble under what appeared to be the diaphragm in elevated posi-

tion, and it was conjectured that this might indicate herniation of the stomach above the diaphragm rather than elevation of the diaphragm (Figure 2).

Three hours after entry the patient regained consciousness and rallied on continuous oxygen and plasma infusion. Nineteen hours after entry he was transferred to a private hospital where, after intratracheal anesthesia and blood transfusion, the left thorax was opened through the eighth interspace. Immediately beneath the incision the traumatized spleen appeared, with the splenic flexure and most of the transverse portion of the colon and the omentum. Several loops of small bowel also lay in the thoracic cavity.

The entire stomach, distended to capacity with food, had rotated 180 degrees to the left, the lower esophagus was angulated, and the greater curvature of the stomach was at the level of the apex of the left lung. The lung was completely atelectatic. The diaphragm, avulsed from sternal and anterior attachments, had a tear in it from the middle of the avulsed border to the posterolateral border, and it was lacerated from the esophageal hiatus to the costal attachment.

The thoracotomy wound was extended downward into a left upper rectus incision, through which the assistant placed his hand to retain the displaced viscera in the abdominal cavity, in order to facilitate repair of the extensive diaphragmatic defect. It was necessary to crush the phrenic nerve. The avulsed border of the diaphragm was then reattached to the anterior thoracic cage about one inch above the original site. This was accomplished by means of interrupted through-and-through mattress sutures of doubled No. 2 chromic catgut, the costal cartilage and ribs being encircled wherever possible.

The long anteroposterior rent was closed with interrupted No. 16 cotton and the suture line "pleuralized" by a continuous over-and-over suture of 00 chromic catgut. The lung was reexpanded, a Pezzar catheter placed for underwater drainage and the wound closed in the usual manner.

Except for the accumulation of 800 cc. of serosanguinous fluid in the right pleural cavity, the postoperative course was uneventful.

The patient returned to his former occupation in two months and has been perfectly well for the two years since. Chest x-rays made at six-month intervals have shown no abnormalities (Figure 3).

501 Professional Building.

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Cytomegalic Inclusion Disease in an Adult

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THE PRESENCE of intranuclear inclusions typical of cytomegalic inclusion disease has been reported observed at autopsy in the salivary glands of as many as 32 per cent of stillborn, newborn and young infants regardless of the cause of death.[3] Smith and Vellios[6] collected reports of 89 cases in infants and children, not including stillborn infants, in which there were more or less generalized inclusions. From a study of those cases, they concluded that (1) in infants under two months of age generalized infection by the salivary gland virus is usually a primary, fatal disease and that the most common manifestations of it are blood dyscrasia and hepatic damage; (2) in children over two months of age the disease is most often associated with another primary disease and plays only a minor role.

Reports of seven cases in adult patients in which intranuclear inclusions occurred in large cells at various points in the alimentary tract from the esophagus to the anus, without evidence of generalized disease, have been collected.[2] There are, however, reports of only four cases of generalized cytomegalic inclusion disease in adults. Following is a report of such a case in an adult who was extensively studied during the last eight months of his life, although as has always been the case the diagnosis was not made until autopsy material was examined.

REPORT OF A CASE

A 42-year-old married white man first noticed slight enlargement of the abdomen in September, 1949. In October, following a slight cold, a persistent hacking, nonproductive cough developed. In late November the patient began having fever, 99° F. in the morning and gradually rising to 102-103° F. in the afternoon, with occasional slight chills. There was severe pain in the shoulders and hips, but none deep in the bones. The patient did not have headache, paresthesias, indigestion or tarry stools, symptoms referable to the urinary tract, epistaxis or purpura. The medical and family history taken at the time of admittance was essentially non-contributory.

In December 1949 the patient was admitted to another hospital with sore throat, slight papillary eruption on the chest anteriorly, diminished breath sounds and rales at the right base, a Grade 3 systolic murmur that was loudest in the left third interspace, leukopenia and an enlargement of the liver. Penicillin was given, then chloramphenicol, without effect on the symptoms.

In early January, 1950, the patient was admitted to still another hospital with the same symptoms. The abdomen was distended, the liver edge was three finger-breadths below the right costal margin, and the spleen, which was soft, was two finger-breadths below the left costal margin.

Roentgenograms revealed a low grade inflammatory process at the base of the right lung, enlargement of the liver and suggestion of inflammatory changes in the ileum.

Results of sternal marrow examination on January 9, 1950, were reported as indicating erythroblastic activity, not diagnostic of leukemia. The cell differential count was reported (per cent):

Blasts	3.5	Eosinophils	2.0
Promyelocytes	5.0	Basophils	0.0
Myelocytes	11.5	Lymphocytes	8.5
Metamyelocytes	1.0	Monocytes	2.5
Non-segmented	26.5	Erythroblasts	8.5
Segmented	4.5	Normoblasts	26.5

Leukocytes in the blood between January 9 and February 13 varied from 1,500 to 4,000 per cu. mm. The proportion of lymphocytes never exceeded 2 per cent, and of monocytes varied from 0 to 28 per cent with most counts closer to the lower figure. Ten transfusions maintained the erythrocyte content at 4 million per cu. mm. of blood.

The patient was transferred to the U. S. Naval Hospital at Oakland on February 17, 1950. Possibly pertinent additions to the history at that time were that he had had blood in nasal secretions occasionally since a youth; that about five weeks before admittance there were two episodes of epistaxis, each lasting about five minutes; and that the skin and sclerae had become icteric in December 1949. The body weight was 20 pounds below the 133-pound usual for the patient.

From the Pathology Service of the U. S. Naval Hospital, Oakland. The statements and conclusions expressed by the authors are their own and do not necessarily reflect the opinion or policy of the Navy Department.

Presented before the Section on Pathology and Bacteriology at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

The blood pressure was 92 mm. of mercury systolic and 55 mm. diastolic, the pulse rate 90, and respirations 18 per minute. Slight icterus, a few scattered petechiae and slight cyanosis of the nail beds were noted. The inguinal lymph nodes were slightly enlarged. The left pupil was larger than the right, but both were regular in outline and normally reactive to light. The tongue was beefy red with moderate papillary atrophy. Increased breath sounds were noted at the bases of the lungs. There was tenderness in the left costovertebral angle. The abdomen was slightly distended and shifting dullness and fluid wave were noted. The edge of the liver was palpated four finger-breadths below the right costal margin. There was slight sacral edema but no pretibial pitting. Results of a neurological examination were within normal limits.

The number of erythrocytes per cu. mm. of blood averaged 3 million; of leukocytes 700 to 900, with 1 to 3 monocytes. The proportion of reticulocytes was from 0.2 to 0.6 per cent. Platelets numbered 340,000 per cu. mm. No pathogenic organisms grew on repeated cultures of blood and urine, no ova or parasites were noted in examination of the stools, and specimens of sputum and gastric secretions contained no acid-fast bacilli. Results of agglutination tests were negative for typhoid, paratyphoid and Brucella. No malignant cells were seen in aspirated biliary fluid. Cephalin-cholesterol flocculation was 3+ in 24 hours. The bilirubin content of the serum was 1.36 mg. per 100 cc. by direct and 4.17 mg. by direct reaction. Prothrombin content was normal. Peritoneal fluid (1400 cc. with specific gravity of 1.010 removed March 7, 1950) showed "inflammatory reaction." In x-ray films of the chest and abdomen, adhesions were noted between the lung and the right leaf of the diaphragm laterally, and peritoneal fluid was present. The urine contained 10 to 12 leukocytes and 40 to 50 erythrocytes per high power field. Erythrocyte fragility was slightly diminished. Biopsy specimens of the liver were removed by needle on March 16 and April 19. The architecture of the specimens was slightly distorted. The few veins present were fairly well preserved and there was no surrounding reaction. In the periportal spaces there was necrosis of parenchymal cells, with infiltration of leukocytes which were lymphocytic, polymorphonuclear and plasma cell in type, with a few fibrocytes. Almost no collagenous connective tissue was present. There was no evidence of regeneration of bile ducts or of liver parenchymal cells. The observations were interpreted as consistent with subacute hepatitis.

Course in Hospital: The patient received a high protein diet with protein hydrolysate, vitamins B_1, B_{12} and K and neostigmine. Blood and salt-free albumin solution were given intravenously. The patient improved subjectively, but there was no objective change other than slight diminution of edema and ascites. The body weight diminished to 93 pounds.

Beginning March 2, corticotropin (ACTH) was administered in a divided daily dose of 40 mg., and beginning April 21 chloramphenicol was given. The temperature became normal and the liver and spleen decreased in size. Chloramphenicol was discontinued May 3, and the following day the dosage of corticotropin was reduced to 20 mg. daily. On May 10 the temperature rose to 101° F. At that time the patient was noted to have a fluid intake of 4500 cc. and an output of 5100 cc. of urine daily. The temperature continued to rise to 105° F. (rectal). The pulse rate rose to 160 and respirations were 36 per minute. The patient died May 11.

AUTOPSY

Macroscopic Observations: Autopsy was performed three hours after death. The skin and sclera were icteric, the mucous membranes pale. The right inguinal lymph nodes were enlarged.

Thoracic Cavity: The mediastinum was not deviated. The pericardium was thickened. There was 50 cc. of clear amber fluid in the pericardial sac. Except that it was small (200 grams), the heart appeared to be normal. There was 50 cc. of clear fluid in the right pleural cavity. The left lung was tightly bound to the lateral chest wall and to the diaphragm. The right lung weighed 800 grams, the left 750 grams, and filled its pleural cavity. The upper lobes were normal in appearance and consistency. Moderate congestion was noted in the hilar regions bilaterally. Thick tenacious mucus exuded from the cut bronchi. The peritracheal and peribronchial lymph nodes were enlarged.

Abdominal Cavity: The diaphragm extended to the level of the fourth rib bilaterally. There was no fat on the omentum. Approximately 200 cc. of bile-tinged fluid was present. The liver weighed 3200 grams. On the surface of the organ, which was smooth, were many grayish areas, sharply demarcated from the surrounding parenchyma. On section these areas had appearance of an infiltrative process of somewhat myxomatous consistency. The portal system and the extrahepatic and intrahepatic biliary ducts were not grossly abnormal. The spleen weighed 975 grams, and on the external and cut surfaces were grayish-white infiltrations similar to those noted in the liver. The pancreas weighed 200 grams and was apparently normal. Each of the adrenal glands weighed 10 grams. The cortices were somewhat thinned and appear devoid of the usual lipid.

The right kidney weighed 245 grams, the left 215 grams. The cortices were 5 mm. thick but were well demarcated from the medullae. There was slight dilation of the pelves and calyces. The ureters, bladder and prostate were normal. The testes and epididymes were small, soft and slightly friable.

No abnormalities were noted in the vascular system, in the muscles (except for slight wasting) or in the skeletal system either externally or on sections.

The brain weighed 1460 grams. Moderate engorgement of superficial vessels was noted. No macroscopic abnormalities were observed in examination of sections.

Gross pathologic diagnoses: Chronic lymphatic leukemia with splenomegaly, hepatomegaly, icterus, pulmonary edema and congestion.

Microscopic Observations

Lungs: The alveoli were filled, but not packed, with leukocytes, largely polymorphonuclear. In addition there were macrophages containing small amounts of brown to black pigment and numerous large cells with pale acidophilic cytoplasm and frequent basophilic areas eccentrically located in a perinuclear manner. The nuclei were large and contained large deeply acidophilic, homogeneous, slightly irregular inclusions up to 7 micra in diameter. These were usually single but occasionally a second smaller inclusion was noted. They were sharply demarcated from the nuclear chromatin, although halo formation was not constant. In a few of these cells there were also small brownish black granules not over 1 micron in greatest dimension in the cytoplasm. Many of these cells were free within the alveoli; others in intimate contact with the alveolar walls (Figure 1). Moderate leukocytic infiltration and minimal dilation of the capillaries were noted in the alveolar walls.

Liver: The general architecture was preserved. The capsule was not thickened. About the portal triads were accumulations of leukocytes, largely lymphocytes. There was necrosis of the parenchymal cells and dilation of the sinusoids, most pronounced in the areas about the central veins.

In the areas of necrosis there was infiltration by polymorphonuclear leukocytes, which were also present in increased numbers within the sinusoids. Approximately half of the parenchymal cells were necrosed. The remainder contained little glycogen and frequent golden granules. There was no apparent increase in fibrous tissue. No evidence of regeneration of parenchymal cells or of duct proliferation was noted. The Kupffer cells were not involved. Foamy macrophages were observed within the sinusoids. An occasional liver parenchymal cell, usually at the periphery of one of the zones of necrosis was approximately twice the diameter of the remaining cells with a proportionately enlarged nucleus containing a deeply staining acidophilic inclusion. These cells were similar to those described in the lung.

Spleen: The capsule was moderately thickened. The trabeculae were widely separated and thickened. The arterioles were diminished in number and widely separated. In those present there was thickening of the walls, with occasional obliteration of lumen. The Malpighian corpuscles were small and without germinal centers. Many contained no arterioles. There was increased fibrous tissue within the pulp as well as a light acidophilic groundwork which obliterated many of the sinusoids. Increased blood pigment within the macrophages was noted, particularly at the periphery of the corpuscles. Numerous small old and recent infarcts were present. Crystal violet stained specimens were negative for amyloid.

Kidneys: There was minimal narrowing of the capillary spaces in the glomeruli. The tubules showed minimal atrophy and contained albuminous fluid within the lumina. A single large cell containing an intranuclear inclusion slightly smaller but otherwise identical with those described in the lung, was noted within the lumen of a tubule.

Lymph nodes: There was destruction of the normal architecture. Much of the node was replaced by a homogeneous, lightly acidophilic ground substance similar to that in the spleen. Germinal centers were not noted. The sinusoids, where present, were distorted. In addition to moderately pleomorphic cells of the lymphocytic series, there were a moderate number of large, slightly irregular, lightly acidophilic cells, approximately 50 micra in diameter. No inclusions were noted in them.

Adrenal glands: Except for almost complete absence of cortical lipid, no significant abnormalities were observed.

Vertebral marrow: The fluid was moderately cellular. A few reticulum cells containing indented or even double nuclei were present. The number of megakaryocytes was moderately increased. In the sinusoids there were numerous degenerated cells and masses of homogeneous lightly metachromatic staining material which appeared to be degenerated cells. There were almost no lymphocytes. No inclusions are seen in any marrow cells.

The biopsy material previously taken by needle from the liver was reexamined. In the material obtained April 19 (22 days before death) homogeneous bright acidophilic intranuclear masses up to 7 micra in diameter were observed in many of the parenchymal cells. The larger masses were surrounded by a clear halo separating them from the remainder of the basophilic stained nuclear material. The nuclei and cytoplasm of the latter cells were moderately increased in size and, while intracytoplasmic inclusions were not seen, many contained golden granules within the cytoplasm. In the material obtained March 16, an occasional acidophilic centrally located intranuclear mass up to 3 micra in diameter was noted, but, even in retrospect, such masses could not be identified with certainty as inclusions.

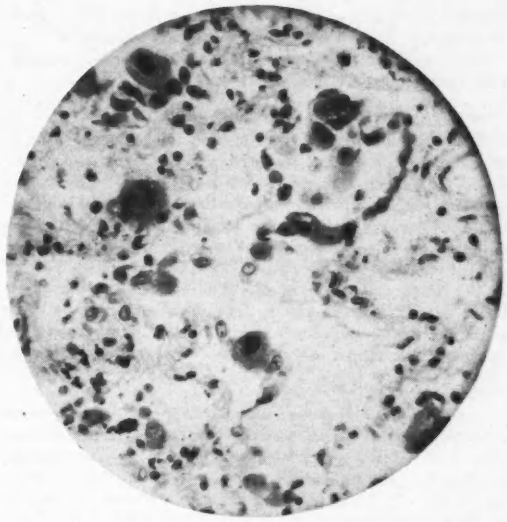

Figure 1.—Intranuclear inclusions in enlarged cells attached to alveolar walls in the lung (x 280).

The inflammatory changes in the ileum, noted roentgenographically five months before death but not at autopsy, may indicate the mode of entrance of the virus in adults in view of reports of these inclusions in biopsy material from various sites in the alimentary tract.[2]

Von Glahn and Pappenheimer's[7] review of the literature with regard to this condition has not been appreciably altered. First considered as protozoan parasites or saprophytes, later as an alteration in cells due to various diseases, these inclusions are now generally considered due to infection by a specific virus. Similar rodent infections may be transmitted by intracerebral inoculation, but species specificity is, to date, absolute. All attempts at animal or yolk sac passage of human virus have failed.

Farber and Wolbach[1] in a series of 183 autopsies on infants who had died of various causes demonstrated the presence of such inclusions within the duct epithelium of the salivary glands of 12 per cent of the subjects. They presented the view that, although due to a specific virus, which may be transmitted transplacentally, these inclusions are not related to illness or death. McMillan,[3] however, expressed belief that the virus causes a disease which is benign unless it involves the lungs where it may give rise to pneumonitis which, if not the cause of death, at least appears to be a definite contributing factor.

DIFFERENTIAL DIAGNOSIS

The large size of the cells whose nuclei contain these spectacular intranuclear inclusions generally occurring in the salivary glands, lungs, kidneys, intestines and spleen, would appear to make oversight almost impossible. In addition, smaller and frequently overlooked cytoplasmic inclusions are present in many of these same cells, apparently developing about seven days after the appearance of the intranuclear inclusions.[3] The intranuclear inclusions are bright acidophilic when stained by hematoxylin-eosin, red when Masson's trichrome is used, and black when the staining material is iron hematoxylin. They do not take up McManus stain. The cytoplasmic inclusions are variously

described as being generally 1 micron or from 2 to 4 micra in diameter. They are basophilic and not vacuolated.

Brief notes on the four other recorded cases of generalized cytomegalic inclusions in adults follow:

Case of Von Glahn and Pappenheimer[7]

A 36-year-old white man had non-amebic liver abscess, and fever for a period of three and one-half weeks. The autopsy diagnosis was abscess of the liver, ulcerative colitis (cecum) with hemorrhage, suppurative pleurisy and sclerosis of the pulmonary venules. The intranuclear inclusions were noted in cells in capillaries and connective tissue throughout the wall of the liver abscess, in the ulcers in the cecum, in the lining cells of some of the alveoli and within capillaries in the adrenal glands. These authors did not note intracytoplasmic inclusions nor did they note cytoplasmic degenerative changes other than the occasional fat globules.

Case of McMillan[3]

A 60-year-old Japanese woman, 32 years a resident of Canada, died of pneumonitis. The patient also had had vitamin deficiency which had responded rapidly to therapy. Cytoplasmic and nuclear inclusions were noted in the cells lining the alveoli of the lungs and in the zona reticularis of the adrenal cortex.

Case of Reinhard and co-workers[5]

The case was reported incidentally in a discussion of the chemotherapy of malignant disease. The patient was a 49-year-old man with subleukemic lymphatic leukemia who had been treated by amino-an-fol, a folic acid antagonist. At autopsy no remainder of leukemia could be identified but foci of giant cells containing the typical intranuclear inclusions were observed in the lungs, spleen and liver.

Case of Wyatt and co-workers[8]

A 27-year-old woman died after seven years of illness that was characterized by aplastic anemia and atypical subleukemic myeloid leukemia. Hypoplastic bone marrow and transfusional siderosis were noted at autopsy. The liver and pancreas were large, firm and dark brown. Inclusions were noted in the liver, lungs, adrenal glands and pancreas. However, the lungs showed no inflammatory reaction. The diagnosis of leukemia was not substantiated by observation of the condition of the marrow at autopsy. The possibility that the virus was transmitted by transfusion to an already debilitated patient and thus became generalized was considered.

That two of the patients in the four cases had leukemia and another had liver damage is of interest with relation to the findings in the present case. With these exceptions, there appear to be no common factors in the clinical courses or pathologic findings.

The majority of all cases, including those in stillborn babies and infants, have been reported from the St. Louis area. This is regarded as a manifestation of particular interest in the condition by pathologists in that area.

SUMMARY

Cytomegalic inclusions were found in the lungs, liver and kidneys of an adult who died of malignant lymphoma.

Undiscovered until examination of the autopsy material, typical intranuclear inclusions were present in material obtained at liver biopsy 22 days prior to death, although inclusions were absent or equivocal in similar material obtained 56 days prior to death.

There was roentgenographic evidence of ileitis four months prior to death. (Gastrointestinal tract lesions containing similar inclusions have been reported in living adults.)

This is the fifth reported case of generalized cytomegalic inclusion disease in an adult and is the third in which there were bizarre blood changes that were diagnosed during life as leukemia. Blood dyscrasias and liver damage are also frequently observed in generalized infections by the salivary gland virus in infants.

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California MEDICINE

For information on preparation of manuscript, see advertising page 2

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EDITORIAL

Veterans and Politics

AMERICA has long recognized the responsibility of government to compensate those persons who are called into military service in time of need. American standards of pay, allowances, leave and other perquisites for military personnel have long been immeasurably higher than those of other countries. Living standards are likewise higher and, in general, everything possible is done to make military service no more onerous than it must be.

Not only is the soldier, sailor or marine well cared for during his term of service; he is well provided for on his return to civilian life. Severance allowances take care of his immediate cash needs; special laws have been passed to allow him to acquire an education at government expense. He may purchase surplus government equipment and buy a home on a priority basis; he makes a lower down payment and receives more government assistance in the purchase of a home than does the ordinary citizen.

All this comes about whether the ex-serviceman has served for 30 days or 30 years. As a veteran, combat or not, he falls heir to the many advantages which a kindly and appreciative government has provided as restitution to him for time spent serving his country.

Since the close of World War I a large part of this governmental compensation has come in the form of medical benefits. The veteran with a service-connected disability has long been entitled to medical and hospital care under the program of the Veterans Administration. Since the close of World War II, the veteran in many states has been allowed to secure his medical care in his own home town from his own physician, an arrangement which eliminates the need to travel to Veterans Administration hospitals and to remain away from his family. A less obvious but quite real benefit is that of avoidance of costly stay in a VA hospital, which must be met out of the taxes the veteran and his fellow citizens pay.

California has been one of the leaders in providing its veterans with home-town medical care. A contract between the Veterans Administration and California Physicians' Service has operated with amazing smoothness and efficiency for several years, with satisfaction to veterans, to physicians and to the government.

When the Congress, last October, saw fit to reduce the overall budget for the Veterans Administration by $40,000,000, the VA applied about three-fourths of the reduction to its medical program. A part of this reduction was made in the home-town medical care plan, with the result that the California program was reduced by about 30 per cent.

With this cut, many of the 11,000 California veterans receiving treatment under the home-town program found their care reduced. Some of them found that their three visits weekly or monthly to the doctor were cut to two. Others continued their full treatment and paid the difference in fees themselves. In other cases the doctors donated their services for the visits eliminated under this economy program.

Investigation of this situation has developed the fact that there has apparently been an application of economy in the direction of producing a public protest, rather than in a way to eliminate expendable services. As one California Congressman wrote, ". . . unfortunately, when Congress attempts to reduce expenditures and balance the budget, some administrative agencies apply the reductions where they will produce the loudest public squawk, not where they will eliminate useless positions and services."

The California Medical Association, in common with other medical organizations, has long supported the policy of providing adequately for the military

veteran who has incurred a disability in the course of his military service. The Association has long decried the policy of extending *ad infinitum* the numerous governmental handouts to all veterans and their dependents. The serviceman wounded in battle or otherwise disabled in service is entitled to all the benefits of the workman injured in the course of his employment and granted care and restitution for his injury. On the other hand, the fact that a citizen was drafted and wore his country's uniform for 30 days should not entitle him to benefits out of all proportion to the contribution he has made to his country.

In the present instance, it is obvious that an economy-minded government will seek to eliminate waste in the Veterans Administration and in all government departments. If the various departments apply their economy hatchets to essential services and produce a public outcry, no benefit will result from the overall economy movement. On the other hand, if the government agencies seek out waste and inefficiency, we may look for a continuation of needed services and a tax reduction simultaneously.

The veteran with service-connected disability needs and should have medical care. The home-town program is supplying that care with efficiency and economy. Let us hope that this one department of the VA may be restored to its rightful place in the program and that fiscal savings be effected in other departments not bearing so closely on the lives and the health of those disabled in the service of the country.

LETTERS to the Editor . . .

Carcinogenic Cigarettes

SINCE it is difficult to obtain convincing statistical evidence for or against the alleged carcinogenic action of cigarette smoke, recent investigators have turned to animal experimentation. Lorenz and his associates, for example, exposed strain "A" mice to tobacco smoke in an especially designed smoking machine for 28 to 250 days. Strain "A" mice have an hereditary tendency to develop lung tumors. They reported that no lung tumors were induced by the tobacco smoke, the number of pulmonary neoplasms being the same in the smoked and control mice.

Recently Essenberg* of the Department of Anatomy, Chicago Medical School, repeated these experiments, subjecting strain "A" mice to a more prolonged exposure to cigarette smoke. He devised a rotary cigarette carriage holding 12 cigarettes (brand name not given). An electric cigarette lighter lighted one cigarette per hour. A vacuum pump created suction just sufficient to burn the cigarette and circulate the smoke and fresh air through the chambers containing the experimental and control animals. The experimental chamber was filled with cigarette smoke for a period of about nine minutes per hour during a 12-hour day for a period of one year.

In a typical test, a group of 36 strain "A" mice was placed in the smoke chamber and the same number in the control chamber. The sexes were equally divided in both chambers. Records of the weight increase and reproductive capacity were kept of all animals. At the end of one year the lungs of each animal were sectioned serially.

In the control group 59.4 per cent of the lungs showed spontaneous tumors, papillary adenocarcinoma being the most common. In the smoked mice 91.3 per cent showed pulmonary neoplasms. The conclusion was drawn that repeated inhalation of cigarette smoke for a period of one year increased by about 50 per cent the tendency of strain "A" mice to develop lung tumors. Whether or not cigarette smoke would have any effect on non-carcinophilic mice has not been determined.

Other deleterious effects were noted in the smoked mice, degenerative changes being recorded in the endocrine glands, kidney, liver, and organs of reproduction. Weight records showed that the smoked mice grew more slowly and failed to attain the full weight of the controls. No young were obtained from the smoked mice. The control mice reproduced freely. Atrophy of the reproductive function was thus one of the outstanding deleterious effects.

W. H. MANWARING, M.D.
Palo Alto, Calif.

*Essenberg, J. M.: Cigarette Smoke and the Incidence of Primary Neoplasm of the Lungs in the Albino Mouse, Science, 116:561, Nov. 21, 1952.

California MEDICAL ASSOCIATION

NOTICES & REPORTS

Executive Committee Minutes

Tentative Draft: Minutes of the 236th Meeting of the Executive Committee, San Francisco, February 1, 1953.

The meeting was called to order by Chairman Lum in Room 221 of the St. Francis Hotel, San Francisco, at 1:15 p.m., Sunday, February 1, 1953.

Roll Call:

Present were President-Elect Green, Council Chairman Shipman, Auditing Committee Chairman Lum, Secretary Daniels and Editor Wilbur. Absent for cause, President Alesen and Speaker Charnock.

A quorum present and acting.

Present by invitation were Executive Secretary Hunton, Mr. George Smith of legal counsel, Councilor Arthur A. Kirchner, Mr. Charles O. Finley, insurance broker, and Mr. Patrick McIntyre, representing Continental Casualty Co.

1. *Proposed Group Accident and Health Insurance:*

Doctor Kirchner and Messrs. Finley and McIntyre discussed the features of a proposed group accident and health insurance coverage for members of the Association. It was pointed out that with a 50 per cent participation of eligible members, all applicants during the charter enrollment period would be accepted regardless of physical history; thereafter, new members of the Association would be enrolled on the same basis but new applicants who had been eligible to enroll in the charter period would be accepted only on the basis of a medical screening.

Discussion was held as to the possibility of extending the period of disability benefits from two to five years for disability arising from illness and Mr. Finley stated this could be done. Benefits for disability arising from accidents would be for an indefinite period. It was pointed out that a favorable loss experience would result in an increase in benefits.

The Association would be required, under state law, to serve as the collecting agency and would furnish the mailing list of present and new members. An enrollment fee would cover the Association's expenses.

On motion duly made and seconded, it was voted to submit this proposal to an independent insurance analyst and to explore the possibility of alternative offerings.

2. *Central Office Lease:*

On motion duly made and seconded, it was voted to confirm an earlier telephone vote approving the signing of a five-year lease on enlarged office quarters at 450 Sutter Street, San Francisco.

3. *Physician Placement:*

A suggestion from Councilor Carey, calling for a review of hospital interns and residents as potential physicians for rural communities was discussed and on motion duly made and seconded, the executive secretary was instructed to follow up on this idea and to add the county medical societies as a further source of names of physicians who might serve in rural areas.

4. *Additional Delegate to A.M.A.:*

Mr. Hunton reported that the Association had received confirmation of an additional delegate to the American Medical Association, effective with the 1953 sessions. An election to this office, subject to official confirmation of the post by the A.M.A., was held at the 1952 Interim Session of the House of Delegates.

5. *State Department of Public Health:*

A request from the State Department of Public Health, asking that the Committee on Public Health and Public Agencies be authorized to seek additional consultation on specific problems, was discussed. It was pointed out that the coming distribution of immune gobulin would call for special knowledge of the problems involved. On motion duly made and seconded, it was voted to authorize the committee to seek additional counsel on the problem of immune globulin distribution.

Adjournment:

There being no further business to come before it, the meeting was adjourned at 4:45 p.m.

DONALD D. LUM, M.D., *Chairman*
ALBERT C. DANIELS, M.D., *Secretary*

In Memoriam

BRAY, EULYS W. Died in Altadena, January 27, 1953, aged 43. Graduate of the College of Medical Evangelists, Loma Linda-Los Angeles, 1938. Licensed in California in 1938. Dr. Bray was a member of the Stanislaus County Medical Society, the California Medical Association, and the American Medical Association.

✤

CIERI, JOSEPH D. Died in Oakland, January 26, 1953, aged 51, of coronary occlusion. Graduate of the Washington University School of Medicine, St. Louis, Mo., 1931. Licensed in California in 1942. Dr. Cieri was a member of the Alameda-Contra Costa Medical Association, the California Medical Association, and the American Medical Association.

✤

CREASE, FREDERICK J. Died in Bakersfield, November 19, 1952, aged 84, of myocarditis, gangrene of the left foot, and arteriosclerosis. Graduate of the University of the South Medical Department, Sewanee, Tenn., 1898. Licensed in California in 1901. Dr. Crease was a retired member of the Kern County Medical Society, and the California Medical Association.

✤

LIVINGSTON, WILLIAM R. Died in Oxnard, December 18, 1952, aged 82. Graduate of the University of Illinois College of Medicine, Chicago, 1893. Licensed in California in 1899. Dr. Livingston was a retired member of the Ventura County Medical Society, and the California Medical Association.

LURIE, SOPHIE A. Died in Los Angeles, January 20, 1953, aged 71, of carcinoma of the stomach. Graduate of Kiev Medical Institute, Kiev, Ukrainian S.S.R., 1913. Licensed in California in 1927. Dr. Lurie was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

MCGEE, R. PROCTOR. Died in Los Angeles, January 14, 1953, aged 75. Graduate of the Denver College of Physicians and Surgeons, Colorado, 1903. Licensed in California in 1925. Dr. McGee was a retired member of the Los Angeles County Medical Association, and the California Medical Association.

✤

TURNER, JAMES H. Died in Lynwood, January 21, 1953, aged 74, of congestive heart failure. Graduate of Tufts College Medical School, Boston, Mass., 1903. Licensed in California in 1914. Dr. Turner was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

WILLISON, EUGENE E. Died in Merced, January 24, 1953, aged 54, of coronary artery disease. Graduate of the Indiana University School of Medicine, Bloomington-Indianapolis, 1927. Licensed in California in 1937. Dr. Willison was a member of the Merced County Medical Society, the California Medical Association, and the American Medical Association.

NEWS & NOTES

NATIONAL · STATE · COUNTY

ALAMEDA

Examinations for certification by the **American Board of Preventive Medicine** will be held April 23, 24 and 25 at the School of Public Health, University of California, Berkeley. Examinations may be taken later in the year at New York City at the time of the annual meeting of the American Public Health Association, November 7, 8 and 9. Persons wishing to take the examinations are required to notify the board, 615 North Wolfe Street, Baltimore.

LOS ANGELES

A two-day sectional meeting of the **American College of Surgeons**, in joint sessions with the Southern California chapter of the college, will be held at the Hotel Statler, Los Angeles, on March 30 and 31. This is the first sectional meeting in Los Angeles since 1946. Dr. Ewing L. Turner, assistant clinical professor of surgery, University of California School of Medicine, Los Angeles, is chairman of the local committee on arrangements. Dr. Clarence E. Rees of San Diego is president of the Southern California chapter of the college. Of special interest at these meetings will be ophthalmological and otolaryngological sessions, in addition to those on general surgery, the announcement of the meeting said.

* * *

The Metropolitan Dermatological Society of Los Angeles has elected the following officers for the year 1953: President, Dr. Stanton B. May, Glendale; vice-president, Dr. Fred F. Feldman, Beverly Hills; secretary-treasurer, Dr. Irving A. Lewe, Montebello.

* * *

Dr. John D. Camp of Los Angeles recently was elected president of the American College of Radiology, succeeding Dr. John S. Bouslog of Denver. **Dr. Earl R. Miller** of San Francisco was elected to the board of chancellors of the college.

* * *

The Third Biennial **Western Conference on Anesthesiology** will be held at the Ambassador Hotel, April 8, 9 and 10.

The conference is sponsored by the California Society of Anesthesiologists and the Northwest Society of Anesthesiologists. As in the preceding two conferences, the program has been constructed about one main subject—this year, The Nervous System.

* * *

More than 2,000 industrial physicians, dentists, nurses and hygienists will meet in Los Angeles April 18 to 24 for the 1953 National Industrial Health Conference.

Members of six national organizations will attend the sessions: American Conference of Government Industrial Hygienists, United States Navy Industrial Health Organization, American Association of Industrial Dentists, American Industrial Hygiene Association, Industrial Medical Association, and the American Association of Industrial Nurses.

This will be the first meeting of the groups west of the Mississippi; previous sessions have been held in the more highly industrialized sections of the East and Mid-West.

Subjects will range from technical discussions of specific industrial health problems to such as the air pollution prevention and control problems in urban industrial centers.

Scores of widely recognized authorities from all parts of the country will participate in the program.

* * *

Elected to office at a recent meeting of the newly organized **Southern California Psychiatric Society,** a district branch of the American Psychiatric Association, were: President, Dr. Mathew Ross; president-elect, Dr. Charles W. Tidd; secretary, Dr. Jerome M. Kummer; treasurer, Dr. Leo Rangell.

Councilors, for terms expiring 1956: Drs. Allen J. Enelow, Samuel Futterman, Jack B. Lomas, and Harry Nierenberg; for terms expiring 1955, Drs. Ralph R. Greenson, Judd Marmor, Robert E. Wyers, and Eugene Ziskind; for terms expiring 1954, Drs. Roberta Crutcher, Norman A. Levy, Clarence W. Olsen, and Eugene Pumpian-Mindlin.

SAN FRANCISCO

Dr. William A. Reilly, director of the Radioisotope Unit at Fort Miley in San Francisco, has been appointed clinical professor of pediatrics at the University of California.

* * *

Opening of San Francisco's newest **arthritis clinic**—at St. Mary's Hospital—was announced recently by Harold W. Knowles, president of the San Francisco Arthritis and Rheumatism Foundation. The new clinic is the fifth of its kind in San Francisco. The others are at the University of California Hospital, Stanford Hospital, San Francisco Polyclinic, and Mount Zion Hospital. All of them are benefited through the Arthritis and Rheumatism Foundation.

GENERAL

The American National Red Cross has announced that upon request made last November by the Office of Defense Mobilization, it will expand its blood collections "to produce as much **gamma globulin** for all purposes as blood processing laboratories can turn out." The announcement called attention to recent experiments indicating that gamma globulin protects against the paralyzing effect of poliomyelitis for a period of about one to five weeks. The Red Cross said it will not allocate or distribute the globulin.

* * *

The **Pacific Dermatologic Association** will hold its 1953 meeting at the Olympic Hotel and University of Washington in Seattle, Wash., on July 9 and 10, 1953. Dr. Earl Osborne, of Buffalo, N. Y., will be the principal guest speaker and will lead symposia on "Cutaneous Malignancy" and on "Industrial Dermatoses." Other features will include luncheon round table discussion groups, a clinical meeting and a histopathologic seminar. Non-member dermatologists are welcome to attend the meeting.

* * *

The seventh annual **Rocky Mountain Cancer Conference** will be held in Denver on July 8 and 9. As in previous years there will be eight outstanding guest speakers, and on

the first evening a banquet and entertainment for physicians and their ladies. There is no registration fee for the conference.

* * *

The 1953 meeting of the **American Goiter Association** will be held in the Drake Hotel, Chicago, May 7, 8 and 9, 1953. The program for the three-day meeting will consist of papers and discussions dealing with goiter and other diseases of the thyroid gland.

POSTGRADUATE EDUCATION NOTICES

MEDICAL EXTENSION UNIVERSITY OF CALIFORNIA

Postgraduate Courses for 1953

Symposia on Psychosomatic Medicine, Wednesday afternoons and evenings, March 11, 18, 25. Fee to be announced. Langley Porter Clinic, San Francisco.

Diagnostic Radiology, April 6, 7, 8, at Franklin Hospital, San Francisco. Fee to be announced.

Pediatric Conference, June 22 through 26. Fee to be announced. Medical Center.

Conference on General Surgery, June 15 through 19. Fee $75.00. Medical Center.

Obstetrical and Gynecological Conference, September 2, 3, 4. Place and fee to be announced.

Ophthalmology (for specialists), September 14 through 19. Fee $75.00. Medical Center.

Medicine for General Practitioners, September through November. East Oakland Hospital. Fee $50.00.

Evening Lectures in Medicine, September through November. Fee $50.00. Mills Memorial Hospital, San Mateo (probably).

Contact: All inquiries to be addressed to Stacy R. Mettier, M.D., Professor of Medicine, Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22.

STANFORD UNIVERSITY SCHOOL OF MEDICINE

The Stanford University School of Medicine will offer the annual postgraduate conference in Clinical Ophthalmology from March 23 through 27, 1953. The program this year will be devoted to Ophthalmic Surgery.

Registration will be open to physicians who limit their practice to the treatment of diseases of the eye or eye, ear, nose and throat. In order to allow free discussion by members of the conference, registration will be limited to thirty physicians.

Instructors will be Dr. A. Edward Maumenee, Dr. Dohrmann K. Pischel, Dr. Jerome W. Bettman, Dr. Max Fine, Dr. Earle H. McBain, and Dr. Arthur J. Jampolsky.

Cardiology—Date: June 15-19. Fee: $75.00.

General Medicine—Date: June 15-19. Fee: $75.00.

Surgery of Trauma—Date: June 22-26. Fee: $75.00.

General Surgery—Date: June 22-26. Fee: $75.00.

Programs and further information may be obtained from the Office of the Dean, Stanford University School of Medicine, 2398 Sacramento Street, San Francisco 15, California.

UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE

Division of Medical Extension Education

No. 882—Essential Physics in Radiology

Dates: March 9, 1953, through April 10, 1953—Los Angeles County Hospital; April 13, 1953 through May 25, 1953—Cedars of Lebanon Hospital.

Tuition: $55.00.

Speakers: Robert E. Pugh, Jr., F.A.C.R. (Assoc.), Henry L. Jaffe, M.D.

Contact: Dr. Gordon E. Goodhart, Director, Medical Extension Education, 1200 North State Street, Los Angeles 33, Calif., CApital 4195.

INFORMATION

Nursing Homes:

An Analysis of the Types of Patients and the Nursing Services

BERNICE HOTCHKISS, R.N., *Hospital Nursing Consultant, Bureau of Hospitals, California State Department of Public Health*

INTRODUCTION

THE BUREAU OF HOSPITALS of the California State Department of Public Health in the administration of the hospital licensing program has felt a need for more information regarding the services which are available in the nursing homes throughout the state as well as the types of patients which are being cared for in such facilities.

Much is being said about chronic care accommodations and the inadequacies of the present facilities. Hospitals are said to be overcrowded because of the increase of chronic diseases brought about by the extended life span of today's population. It is well known that many individuals cannot be cared for in their homes and are finding their way into "nursing homes." The many inquiries coming to the Bureau of Hospitals are evidence that need exists in many communities for this type of facility.

A study already completed by the State Department of Public Health* revealed that almost 9,000 beds are licensed in the nursing homes of California. These beds are found in 450 nursing homes located throughout the state. That study further revealed that while most of these facilities are relatively safe and are capable of providing adequate facilities for patient care, there are many deficiencies which do exist and correction of which would result in improved patient care more in harmony with what is advocated in the care of the chronically ill.

The following study has as its main objectives the determination of the types of patients being accommodated, the utilization of beds in nursing homes as determined by occupancy, and the nursing services which are being rendered in these nursing homes.

This information was secured from results of a questionnaire completed by field personnel of the Bureau of Hospitals at the time of their regular visits to the nursing homes. A three-month period was used to secure this material and the questionnaire was filled out on all nursing homes visited by the field staff. It can be assumed that the sampling secured by this method is fairly typical of all facilities throughout the state.

One hundred sixty-eight nursing homes, representing a total of 3,652 licensed beds, or 42 per cent of all nursing home beds in the state, are included in the study.

Two thousand eight hundred sixty-five persons were housed as patients in these homes at the time the questionnaire was completed.

CAPACITY

The capacity of the nursing home included in this sampling ranged from 3 beds to 88 beds. The following table shows the range of capacity as well as the number and percentage of nursing homes in each range:

Capacity	*Number of Nursing Homes*	*Percentage of Nursing Homes*
2 to 10 beds	51	30%
11 to 25 beds	52	31%
26 to 50 beds	58	35%
51+ beds	7	4%

For purposes of this study, the above classification of range of capacity has been retained. It has been expressed by some authorities that the size of the facility has a direct bearing on the type of care which is provided as well as the quality of service which is available. Apparent differences in this report will be pointed out.

The range of capacity in the sampling is not typical of all facilities licensed by the State Department of Public Health. Actually only 26 per cent of the total number of nursing homes have more than 25 beds; however, 39 per cent of the homes in the sampling have bed capacities greater than 25 beds. Also, 43 per cent of the total facilities have capacities of fewer than 11 beds, but the study included only 30 per cent in this range. While this discrepancy does not alter the general conclusions in this study, it should be kept in mind where distinctive differences appear between the large and small facilities.

OCCUPANCY

Average occupancy of the 168 nursing homes included in the study was 83 per cent, with a median of 86 per cent. Of the total number of facilities in this study, 99 were operating with occupancies greater than 75 per cent. Only 12 were operating with occupancies less than 50 per cent.

The following breakdown serves to show that the size of the facility has a direct relation to its occupancy, the small nursing home tending to operate at a higher occupancy that the large one.

Reprinted from *California's Health*, November 30, 1952.

* See "Nursing, Convalescent, and Rest Homes in California," Bernice Hotchkiss, R.N., *California's Health*, March 15, 1952.

The small nursing homes of 2- to 10-bed capacity were operating with an average of 92 per cent occupancy. Of the 51 homes in this category, 22 facilities, or 43 per cent, were operating at a 100 per cent occupancy.

In the 11- to 25-bed class, the facilities averaged 87 per cent occupancy. Sixteen nursing homes, or 36 per cent of the 52 nursing homes, were operating at full capacity.

In the nursing homes with capacity beyond 26 beds, the occupancy was 73 per cent, with only seven of 65 facilities operating at 100 per cent occupancy.

These high occupancy figures substantiate the idea that a great need exists for this type of hospital facility. These nursing homes are meeting a definite need and are providing a type of care which apparently is not available or possible in the home or the general hospital. On the basis of this sampling, it can be assumed that about 7,500 persons are being cared for by this category of hospitals in the state.

The higher rate of occupancy in small nursing homes in comparison with the larger is significant, and clearly illustrates the existing demand for this type of facility. There are probably many reasons why these homes were almost fully occupied. It should be remembered that almost every nursing home, whether large or small, is attempting to operate for profit. The small nursing home must meet the same minimum standards for state licensure as the large one. This usually entails a rather large expenditure of money in order to comply with physical requirements. As a result, in order to realize a profit, the small nursing home must operate with all beds occupied. In these small facilities, the operating cost is substantially the same whether all beds are occupied or not. Consequently, greater conscious efforts are probably made by the operator to keep the beds full. There is also the contention that the chronically ill patient in the small nursing home can enjoy more of a home environment than is possible in the larger, institutional type of facility.

TYPE OF PATIENTS

In an effort to determine the amount and type of personnel necessary for the care of these patients, some information on the physical status of the patient was obtained. While it is not always true that the bed patient requires more care than the ambulatory patient, it does give some idea as to the attention and care which is usually required. Size of the facility apparently does not affect the types of patients which are accepted for care so the breakdown by size will not be considered in this section of the report.

Of the 2,865 patients in the 168 nursing homes, 5 per cent required no specific care. The services which were provided for them consisted of board, room, and minimum supervision. (These patients are ambulatory, are able to carry on their ordinary routine without assistance and need only the protection of having a responsible person on hand if necessary.) It would appear that this small percentage of persons has been financially able to select this type of facility for everyday living, and these persons probably experience some feeling of security, knowing that residence can be retained in case of illness.

Thirty-six per cent, or 917 patients, were ambulatory but required some assistance and attention. Such assistance may have consisted of help in such daily tasks as bathing, walking or dressing, or it may have meant providing therapeutic diets, administering medications, dressings or treatments. Such attention and care may be necessitated by physical infirmity and senility, by chronic disease, or by convalescence from acute illness. While this type of patient usually does not require as much care as bed patients, some patients in this category do require skilled personnel for medical and nursing care and supervision.

While 59 per cent of the patients included in the study were classified as bed patients, 23 per cent were declared to be absolute bed patients while the remaining 36 per cent could get out of bed or be assisted out of bed at least every day. The patients designated as strict bed patients were unable to be out of bed because of physicians' orders, because of disease or physical infirmity, or because of lack of encouragement and assistance by the staff in the nursing home. Bed patients, whether absolute or not, require extensive and skillful nursing care. Even though a patient is allowed out of bed for a few minutes or a few hours every day, skillful handling and close supervision are usually required. The procedure of helping a patient get out of bed and into a chair often requires as much skillful handling and consumes more time than if the patient were left in bed. In fact, some of the homes reporting a high percentage of strictly bed patients, could probably reduce this percentage if efforts were made to get patients out of bed. Lack of personnel and lack of knowledge of modern care of the chronically ill is probably responsible for this high percentage.

It was noted that 22 nursing homes had no ambulatory patients at all, but listed all their patients as bed patients with some of those bed patients being able to be up in a chair every day.

Thirty-two nursing homes declared no absolute bed patients. Operators claimed that all their patients were either ambulatory or could be out of bed with or without assistance.

Fourteen facilities declared all patients to be ambulatory. However, all 14 had patients who required some type of medical or nursing care.

NURSING PROCEDURES

The study shows that 44 per cent of all patients are able to take tub baths, the remainder requiring bed baths. This means that while more than half are given bed baths, some of those being given tub baths are actually bed patients and require attention and skill in the procedure. It is well recognized that in every hospital much time is consumed in the task of bathing patients. From the number of bed baths reported, it was apparent that one-third of the nursing homes were giving their patients bed baths daily. The questionnaire was not intended to get information for the purpose of evaluating adequacy of nursing care, but instead it attempted to get some data which would determine the amount of time consumed in these tasks.

Twenty per cent of the total number of patients were on special therapeutic diets. Special point was made not to include in this percentage diets which were special only because of the patient's inability to chew, swallow, etc. While the special diets in a nursing home are usually relatively simple, a basic knowledge of nutrition and nutritional needs of the individual patient is required in order that physicians' orders be complied with and adequate diets be provided. There is probably need for much to be done in the field of nutrition for patients in these homes. It is felt that many operators either do not realize what the basic nutritional needs of the aged are, or are more concerned with providing a cheap diet than with having the food served meet nutritional needs.

Thirty per cent of the patients required assistance with eating. In some cases this meant actually feeding the patient because he was too ill, needed encouragement, or was not physically able to feed himself. In other cases, it meant only assisting the patient by cutting meat, buttering bread, etc., so that the patient would be able to feed himself.

Two per cent of the patients had pressure sores. The patients with pressure sores were centralized in 46 nursing homes. One home reported that it had four patients with pressure sores, totaling 15 per cent of its patients. Others for the most part reported single patients in their facilities with this affliction.

An average of 31 per cent of all patients in nursing homes were declared to be incontinent, having no control of urine or feces. This is rather a startling figure and presents a nursing problem of great magnitude. It is well known that incontinence is a common accompaniment of old age and likely to occur among such persons. With expert nursing care and more adequate staffing, it is felt that many of these oldsters would not be incontinent and this percentage might be reduced.

About 68 per cent of the patients were getting some type of medication. The most common mode of administration was by mouth, although hypodermic, intramuscular and intravenous methods were employed in many facilities. Only five nursing homes reported that no patients were receiving medication. Fifty-one of the 168 facilities reported that they had given no hypodermics during the preceding week.

In five of the 168 homes, no tasks which can be considered nursing procedures were being performed; in 18, the only procedure was bed baths; in 15, only bed baths and medications; and in 20, bed baths, medications, and enemas comprised the total skilled nursing procedures.

Except for bed baths and medications, the most common nursing procedure being performed in nursing homes was the administration of enemas. Only 37 homes reported that no enemas had been given during the preceding week. The frequency of this procedure is necessitated by the type of elderly patient likely to be found in the nursing home.

Less than half of the nursing homes had been required to apply surgical dressings during the preceding week. Even in these, the questionnaire revealed that usually only one patient in each home was getting regular dressings. It can be assumed that this procedure is not often employed.

Irrigations (eye, throat, bladder) had been performed in 38 nursing homes. This seems unusually low when it is realized that many elderly male patients frequently have in-dwelling catheters which necessitate bladder irrigations.

Intravenous injections, usually medications, had been given in 19 nursing homes during the preceding week.

The use of the heat lamp for treatment of various ailments was common in 29 nursing homes.

Massage, oxygen therapy, diathermy, physiotherapy, inhalation therapy, and application of compresses were other procedures which were listed as less frequently employed in the nursing homes included in the questionnaire.

CONCLUSION

Our nursing homes, in addition to providing protection and custodial care are providing to some extent a certain type of medical and nursing care. The amount of care varies, in that some nursing homes are staffed and equipped to accept patients requiring very extensive and skilled care while others are not able to do so. The need for this type of facility is evidenced by the high occupancy of many of these nursing homes.

As a result of the information gathered for this study, the following nursing procedures appear necessary to provide the medical and nursing services

which should be available in today's nursing homes. It is quite possible and even desirable that other more complex procedures be performed in certain cases where the staff and equipment are available. However, the following procedures appear to be necessary to provide the necessary minimum services.

1. Personal care of patient
   a. Making patient comfortable
   b. Knowledge of supportive measures to be applied for patient's comfort and happiness
   c. Giving bed baths
   d. Assisting with tub baths
   e. Assisting patients in and out of bed
   f. Care of incontinent patients
   g. Skin care, and nail care
      (1) Prevention of pressure sores
      (2) Treatment of pressure sores
   h. Hair shampoos (in and out of bed)
   i. Care after death
2. Medication
   a. Techniques of administration
      (1) Hypodermic
      (2) Intramuscular
      (3) Oral
      (4) Instillation of eye and nose drops
   b. Knowledge
      (1) Usual dosage
      (2) Signs of overdosage
      (3) Methods of administration
      (4) Effect and reaction
3. Nutrition
   a. Knowledge of nutritional needs
   b. Ability to prepare special diets
   c. Feeding patients
4. Charting—patients' records
5. Recognition, observation and interpretation of symptoms
6. Temperature, pulse, respiration and blood pressure
   a. Techniques
   b. Interpretation
   c. Recording
7. Aseptic technique
   a. Knowledge
   b. Methods of applying sterile dressings
8. Compresses
   a. Techniques
   b. Indications, contraindications
   c. Application of ice bags, hot water bottles, etc.
9. Catheterizations
   a. Techniques
   b. Contraindications
10. Douches
11. Enemas
    a. Types
    b. Techniques
    c. Contraindications
12. Application of heat lamp

THE PHYSICIAN'S *Bookshelf*

NUTRITION AND DIET IN HEALTH AND DISEASE—6th Edition. James S. McLester, M.D., Professor of Medicine Emeritus, University of Alabama; and William J. Darby, M.D., Ph.D., Professor of Biochemistry and Director of the Division of Nutrition, Vanderbilt University. W. B. Saunders Company, Philadelphia, 1952. 710 pages, 14 figures, and 145 tables, $10.00.

McLester and Darby have written a most important textbook of applied therapeutics. The sixth edition establishes this volume as one of the necessary books on the physician's ready reference shelf. The first half of the book is devoted to the physiologic and biochemical background of food substances and discussions basic to an understanding of human ecology and its relations to health and disease. The second part of the book is devoted to a detailed discussion of nutrition in disease with very fine chapters on diabetes, gout, obesity, diseases of the kidney and urinary tract, and the digestive, cardiovascular and endocrine systems. There is a very fine chapter on the importance of nutrition in surgery. The volume is carefully annotated, contains many valuable tables and an excellent bibliography. This book is highly recommended without reservation to student, physician and teacher.

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FUNCTIONAL ENDOCRINOLOGY—From Birth Through Adolescence. Nathan B. Talbot, M.D., Associate Professor of Pediatrics; Edna H. Sobel, M.D., formerly Research Fellow in Pediatrics; Janet W. McArthur, M.D., Instructor in Gynecology; and John D. Crawford, M.D., Instructor in Pediatrics, all at Harvard University. Published for the Commonwealth Fund. Harvard University Press, Cambridge, Mass., 1952. 638 pages, $10.00.

The general substance of this book is concisely described by its full title. The authors introduce the discussion of each endocrine gland with a detailed and single exposition of the hormonal physiology involved. A careful description of methods employed for appraising normal action is also provided. After establishing this background, they present clinical material illustrating hypo- and hyperfunction of each gland. Whenever possible precise methods of therapy are given.

This book is not a recapitulation of known facts but an effort to recognize the mass of material that has been accumulating so rapidly in recent years. For brevity and clarity, the authors deliberately present single hypotheses of debatable subjects and defend their newer concepts with experimental and clinical data drawn, for the most part, from their own wealth of experience. This results in a directness and unity of presentation which makes the text exceedingly readable, informative and provocative.

A single endocrine system is discussed in each chapter. There are tables summarizing data and illustrative case histories when pertinent. In addition, there are numerous schematic drawings, apparently included as pictorial aides for the reader. This reviewer found them tedious. However, they may be omitted without sacrificing the lucidity of the text. The clinician who expects to find readily a discussion of a subject, e.g., dwarfism, will be disappointed. The material is so organized that he must consult the clinical section in each chapter. However, this very organization gives value to the book. Emphasis is placed on the action and interaction of hormones in health and disease which has much vaster application in medicine today than a discussion of simple endocrinopathies.

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PARDON MY SNEEZE. Milton Millman, M.D., Fellow American Academy of Allergists. Millman Books, 1635 India Street, San Diego, 1952.

Much of the information and advice contained in Dr. Millman's book, "Pardon My Sneeze," is accurate and might be helpful to patients. He devotes a large amount of space to a discussion of the elimination diet which would probably aid the patient who is receiving this type of diagnostic care. However, his statement that the skin tests for food are only 50 per cent accurate might be questioned by some allergists who have found them to be considerably more dependable than this figure would indicate.

The latter part of the book contains specific information for the allergic patient which should assist him to recognize and shun most common allergens. Chapter 20 is devoted to a discussion of how to avoid some of the more common miscellaneous allergens, both ingestants and contactants, such as flaxseed, orris root, feathers and animal danders. Chapter 21 goes into considerable detail about common food allergens, where they occur and how to avoid them. Chapters 22 through 27, the final chapters in the book, contain recipes and menus for the allergic patient, together with some advice on the use of these recipes. Such data should be helpful to the allergic patient.

The physical appearance of the book itself is not prepossessing with its paper back and unattractive type. There is a lack of dignity in the presentation of the subject matter and the cartoons with which the book is illustrated, as well as the text itself, take a flippant attitude toward allergy. The subject is poorly presented, the book is repetitious and tiresome and would be more likely to discourage the allergic patient than lift his morale and encourage him to continue proper treatment.

Apparently the book was not carefully edited for grammatical errors as they occur with a fair degree of frequency. On page 39, for example, the following sentence appears, "Most cases, fortunately are worked out relatively rapid." Such errors indicate a lack of care in both the writing and correcting of the manuscript. The bibliography is inadequate with less than a dozen references.

In summary, portions of the book will undoubtedly be an aid to the allergic patient but the manner of presentation indicates haste, lack of care and a flippant attitude in the preparation of the manuscript. The physical appearance is not attractive but this might be accounted for on the basis of present high publishing costs.

1952 YEAR BOOK OF OBSTETRICS AND GYNECOLOGY—Edited by J. P. Greenhill, M.D., F.A.C.S., Professor of Gynecology, Cook County Graduate School of Medicine. The Year Book Publishers, Inc., 200 East Illinois Street, Chicago, 1952. 575 pages, $5.50.

The 1952 Year Book of Obstetrics and Gynecology, edited by J. P. Greenhill, again presents a comprehensive review of the world literature as it applies to this specialized field. The articles abstracted in this volume cover the period July 1951 through June 1952. As usual, the work is well done. The material is well-balanced, 283 pages of the text being devoted to obstetrics and 272 to gynecology. Practicing obstetricians will appreciate the generous portion of the former section allocated to the various aspects of labor and problems associated therewith. Adequate coverage of progress in research in gynecological and obstetrical fields is presented, but is not overdone. The editorial comments are cogent. In general, this current edition of the Year Book continues to merit the high place this publication holds as a reference work.

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OPERATIVE NEUROSURGERY — With Emphasis on Procedures in Trauma—Elisha Stephens Gurdjian, M.D., Professor of Neurosurgery, Wayne University College of Medicine, and John E. Webster, M.D., Assistant Professor of Surgery, Wayne University College of Medicine. The Williams and Wilkins Company, Baltimore, 1952. 422 pages, 129 plates, $10.00.

This is a well-written concise book on neurological surgery. It will serve the general practitioner and general surgeon who are often forced to do some neurosurgical procedures on traumatized patients. The contents of the book include four sections: (1) the head, (2) the spine, (3) the autonomic nervous system, and (4) the peripheral nervous system. The illustrations are abundant but extremely difficult to understand. They consist of semi-diagrammatic line drawings but the contrast in them is poor, and the text itself must be carefully read and studied in an effort to understand the illustrations. The book is essentially devoted to surgical techniques with very little discussion of diagnostic and physiological considerations. The book fills a void in neurosurgical literature, for a concise book on neurosurgical operations has been needed. In the past one has had to refer to individual articles or monographs in order to obtain much of the information in this book. This book can be recommended to young surgeons interested in neurosurgery and to practicing surgeons who must do occasional neurosurgical procedures.

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BACTERIAL AND MYCOTIC INFECTIONS OF MAN—2nd Edition—Edited by Rene J. Dubos, Ph.D., The Rockefeller Institute for Medical Research, J. B. Lippincott Company, Philadelphia, 1952. 885 pages, 98 illustrations, $7.50.

The National Foundation for Infantile Paralysis, Inc., has done doctors a good service by aiding the publication of this useful book so as to bring the cost within reasonable limits. Needless to say, Dr. Dubos has assembled a panel of top authorities to write the various sections. The book is not primarily a clinical treatise; while clinical matters are dealt with, it is primarily a discussion of clinical bacteriology. Typhoid fever, for example, which covered 50 pages in Osler's textbook, here receives two pages, and so forth. Swift's article on the streptococci, one of the best in the volume, occupies about 60 pages; of these streptococcal diseases of man are discussed in 15 pages. But a superb background is developed by the fundamental discussion of streptococci from the biological and clinical bacteriological standpoints.

The volume is finely printed and illustrated and each article is followed by a comprehensive bibliography. It fills a gap between the ordinary textbook of bacteriology and the standard textbook on medicine; at the moderate price it should be within the reach of every doctor and student.

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THE TREATMENT OF INJURIES TO THE NERVOUS SYSTEM—Donald Munro, M.D., F.A.C.S., Surgeon-in-Chief, Department of Neurosurgery, The Boston City Hospital; Associate Professor of Neurosurgery, Boston University School of Medicine; Assistant Professor of Neurosurgery, Harvard Medical School. W. B. Saunders Company, Philadelphia, 1952. 284 pages with 47 figures, $7.50.

This monograph presents in a single volume the thoughts and conclusions arrived at by the author during a professional lifetime in which the treatment of injuries to the central nervous system has been his particular interest. Few men have had or will have Dr. Munro's broad experience in the handling of this type of lesion of the nervous system. Coupled with this opportunity, he has brought to bear an original approach to the handling of traumatic problems which has resulted in many therapeutic developments which are in general use today. The section devoted to the management of the paralyzed bladder is of particular interest, as is also the chapter on rehabilitation.

It should be mentioned that there are some opinions and recommendations expressed here which are controversial. An example of this relates to recommended fluid intake following injury. To many practitioners, the amount suggested will seem large and perhaps not in keeping with current thinking concerning water metabolism and brain edema. The author's enthusiasm for section of the tentorium will also require critical consideration.

In the process of putting to use some of these unique recommendations, surgeons must consider a factor which the author could not stress as much as perhaps it should be emphasized; this is the role of constant personal attention to detail which is of such great importance to the success of his endeavor in this demanding field.

The monograph is written for all who are called upon to deal with injury to the nervous system. As such it will be of most interest to the neurosurgeon. To those of this group who will study it as a whole, thoroughly and critically, it will be a welcome addition to the working library.

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STANDARD VALUES IN BLOOD—Being the first fascicle of a Handbook of Biological Data. Edited by Errett C. Albritton, A.B., M.D., Fry Professor of Physiology, the George Washington University. Prepared under the direction of the Committee on the Handbook of Biological Data, American Institute of Biological Sciences, the National Research Council. W. B. Saunders Company, Philadelphia, 1952. 199 pages, $4.50.

It is impossible in limited space to describe adequately the contents of this volume, which in general consists of many tables containing data pertaining to blood. Great pains were taken to assure presentation of the most reliable values for a wide variety of substances. Mean values are supplemented by ranges whenever possible, and the data have been collected from many animals in addition to man. Sources of information are cited.

Subjects include physical properties, coagulation phenomena, blood groups, hemoglobin, blood and bone marrow cells, and chemical substances. One may find the concentration of valine in mouse plasma, riboflavin in snake blood, or arginase activity in man's erythrocytes. Effective levels of therapeutic agents are given. Tables concerning the effects of radiation on peripheral blood and the changes in stored preserve blood are reminders of our times. The volume belongs in the hands of investigators everywhere.